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A SYMPOSIUM UPON NEPHRITIS.

The Pathology and Classification of Nephritis.

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The morbid conditions of the kidney collectively spoken of as *nephritis* present themselves under conditions that may be conveniently grouped as follows:

1. Those depending upon and occurring in association with well-defined and easily-recognized lesions originating primarily in the kidney or the structures with which it is in relation. In this group may be placed nephrolithiasis, hydronephrosis, pyonephrosis, "surgical kidney," tuberculosis, and the neoplasms.

2. Those depending upon the colonization in the kidney of microöganisms brought from distant primary foci of disease by the circulating blood—pyèmia, miliary tuberculosis, septicemia, smallpox, etc.

3. Those depending upon certain constitutional disturbances of uncertain nature, characterized by general fibrosis of all the organs, with cirrhotic changes in the kidney—arteriocapillary fibrosis, arteriosclerosis, chronic syphilis, gout, lithemia, etc.

4. Those depending upon chronic forms of intoxication with fibrotic changes chiefly located in the kidneys—chronic interstitial nephritis, lead poisoning, etc.

5. Those depending upon the action of definite chemical intoxications, as poisoning by cantharides, turpentine, mercuric chloride, phosphorus, ether, etc.

6. Those resulting from intoxications associated with various well-known infectious diseases, such as yellow fever, scarlatina, pneumonia, diphtheria, etc.

7. Those occurring in certain definitely toxic affections not known to be infectious, as acute yellow atrophy of the liver.

8. Those developing during periods when the metabolic and physiologic equilibrium of the body are disturbed, as at puberty, during pregnancy, etc.

9. Those arising from causes not yet explained.

To the writer all nephritis is secondary and toxic. The primary lesions are to be looked for in other organs through whose morbid activities the blood becomes surcharged with matters injurious to the renal tissue.

The studies of toxins made during recent years show us various facts that bear aptly upon the study of nephritis. Thus we find that many toxins have effects, mostly irritative effects, in common; others effects that are peculiar and specific. Toxins of various kinds therefore produce varying effects upon the kidney according to their constitution. Furthermore, toxins not infrequently have definite specific affinities for certain cell groups.

Again, the degree of concentration of the toxin must materially modify the effect it exerts, and finally the duration of the injurious action produces a considerable variation in its effect.

In the study of nephritis we deal mostly with toxic substances that have in common

an injurious effect upon epithelial tissues, and in particular a destructive effect upon the kidney. This is probably because the cells of the kidney being the most active eliminative cells of the body, seize most energetically upon the poisons in order to remove them from the blood. In most cases of nephritis, some parenchymatous degeneration takes place in the tissues of other organs, notably the liver—another great emunctory.

When we come to inquire the actual nature of the toxic agents in nephritis, we find them too numerous, and probably too dissimilar, to permit of intelligent consideration. Some are inorganic, some organic; some are well known to chemistry, some well known to bacteriology, some only conjectural in nature. Some show a specific affinity for the parenchymatous tissues, which they rapidly destroy; some act insidiously but destructively; some gently stimulate the connective tissues to proliferation. The effect of some is shown with great rapidity, that of others very slowly.

Intoxication is rarely a simple process. Where one toxic substance is at work, others soon make their appearance, and several dissimilarly acting poisons are soon coöperating. This affords one explanation of what is well known to every morbid anatomist, that renal disease instead of being a simple process is almost always a complicated one.

Another important fact concerning the action of toxins is their tendency to lack uniformity in their effect upon the tissue and occasion scattered foci of intense action throughout a tissue otherwise but slightly altered. Such focal lesions are seen in poisoning by ricin and by diphtheria toxin, and in the liver in typhoid fever. In lead poisoning minute hemorrhages result from injury to the capillary endothelium, and in other cases where the local action of the toxin cannot be seen, its particular affinity for certain cells and tissues can readily be determined by its physiological effects, as in tetanus and venom poisoning. The application of these effects to the lesions of the kidney will be made clear when we come to consider the morbid appearances presented by the organ in the various forms of nephritis.

Classification.—When we come to the question of classification the complicated nature of nephritis becomes a source of embarrassment, for while there are several well-defined types, from the standpoint of the morbid anatomist, they are rather rarely observed. The great majority of kidney lesions are mixed affections of the stroma and parenchyma.

In the opinion of the writer the more simple the classification of nephritis the better. Several well-defined types exist:

1. Those chiefly affecting the connective tissue framework—*interstitial.*

2. Those chiefly affecting the epithelial tissue—*parenchymatous.*

Each of these may be acute or chronic. Cases that are not distinctly acute or chronic may for convenience be called subacute. How much more particularly the clinician should attempt to analyze the kidney lesions, or what practical advantage there may be in so doing, I do not know, but the writings upon the subject contain a number of varieties which when carefully compared are for the most part synonymous. A few terms, such as *glomerulo-nephritis,* with unusually destructive glomerular changes; *productive nephritis,* with more permanent destructive changes in the glomerules; *diffuse nephritis,* which is the real condition found in most diseased kidneys, except those from the very young, and indicates that the morbid process affects both the connective and epithelial tissues; *desquamative nephritis,* in which the epithelial cells become loosened from the basement membrane and are washed out of the tubules, may be useful to facilitate accurate description, but not as indicating definite forms of the disease.

Other terms are simply cumbersome. Thus *tubular nephritis* cannot in any sense differ from parenchymatous nephritis, since there is no parenchyma apart from the tubules, unless the glomerules are meant, when we already have the term glomerulo-nephritis by which to describe

the condition. It seems equally useless to employ the term *exudative,* when all acute forms of nephritis are exudative, or to speak of *croupous nephritis* simply because there are many cylinders—especially as the cylinders are not formed of fibrin.

Morbid Anatomy.—How far it is wise in this symposium to enter upon the description of diseased kidneys I am uncertain. It seems to me that every one present must be familiar with the purulent excavations of surgical kidney and the abscesses associated with nephrolithiasis which constitute the *acute interstitial nephritis,* and with the little, hard, red, granular kidney with the tightly adhering capsule that characterizes *chronic interstitial nephritis.* I commonly find, however, that the atrophic kidney of arteriosclerosis is often confused with chronic interstitial nephritis, and I feel it necessary to point out that when the kidney is but little below normal size, is deeply pitted and grooved, and permits the capsule to be easily stripped from a smooth, shining surface, it is the former and not the latter lesion that is at hand.

I also feel it unnecessary to describe the enlarged, dark-colored, slightly softened, juicy, bloody organ from which the capsule easily strips, that characterizes *acute parenchymatous nephritis* in its typical form, or the large, pale, grayish or slightly yellowish, firm, inelastic organ with slightly adherent capsule and more or less granular surface characteristic of chronic parenchymatous nephritis. I must, however, be particular to point out that when one sees these typical lesions once he sees the atypical or combined lesions ten times, and that it is usually with some hesitation that a diseased kidney can be assigned to its proper generic place.

When we turn from the macroscopic to the microscopic changes characteristic of nephritis, probably one of the first surprises that we receive is the discovery that the extent of the one is not indicative of the other. A kidney sometimes appears to the naked eye to be seriously and extensively diseased though the microscope reveals but little destruction of the epithelium, and *vice versa* it may appear to be in fair condition when examined with the eye when the microscope shows scarcely any normal epithelium. Another matter of practical importance is the distribution of the morbid change. I think that a careful examination of microscopic sections of diseased kidneys will show that it is the exception and not the rule for the parenchyma to be uniformly affected. If the disturbance affect localized areas of kidney substance only, the urine may show as many indications of disease as if the whole kidney were diseased, but the chances of recovery are in proportion to the extent of normal or fairly normal tissue that remains. A kidney in which some portions remain unaffected while others are seriously diseased is probably a much better organ than one in which all of the substance is but slightly affected. This enables us to understand why some cases with very bad urinary findings recover, while others with fewer signs of disease die. That we possess vastly more renal tissue than is necessary to maintain life is shown by the comparatively good health enjoyed by many whose one kidney has been surgically removed, and the surprisingly small quantity of renal tissue that may support life is demonstrable by experimentally removing from one of the lower animals one entire kidney and a considerable part of its fellow. It must also have been remarked by all who are familiar with hydronephrosis, cystic, tuberculous, and other renal lesions.

The *microscopic* lesions of nephritis affect the *interstitial tissue,* the *glomerules,* and the *tubules.*

(*A*) The *interstitial tissue* shows change in all forms of nephritis, whether interstitial or glomerular. In the forms commonly described as parenchymatous they are usually very simple, and consist of congestion with some edema. It is this congestion and edema that give the increased size, the flabby texture, and the dark color to the diseased organs. In nephritis accompanying certain infectious

diseases, particularly diphtheria, there may be added to the interstitial edema more or less pronounced round-cell infiltration in which polymorphonuclear leucocytes predominate, but in which lymphocytes and plasma cells may be conspicuous. Councilman and his associates have dwelt upon the frequent occurrence of plasma cells in the nephritis accompanying diphtheria. In cases in which microörganisms play a part in the kidney lesions, the round-cell aggregations may be numerous and extensive and even progress to actual suppuration. In chronic parenchymatous nephritis there is usually some edema, but a new factor, that of connective tissue proliferation, makes its appearance, and in protracted cases of chronic parenchymatous nephritis so much new connective tissue may be present as to leave one in doubt whether the primary change was in the connective tissue or in the parenchyma.

In the chronic forms of nephritis commonly called *chronic interstitial nephritis* the new formation of connective tissue predominates over all other changes. It is almost always irregular in distribution, beginning in certain foci and slowly invading the organ, so that early changes of this kind are often described as *focal chronic interstitial nephritis*. In the badly diseased areas the parenchyma—both glomerules and tubules—are markedly atrophic, but between these the tissue may show but little change, and side by side with glomerules so atrophic as to be scarcely recognizable, large, healthy, perhaps even hypertrophic glomerules may be conspicuous objects. In all kidneys of fatal chronic interstitial nephritis some healthy glomerules and tubules can be found. This explains why patients suffering from the disease live so long—until not enough normal tissue is present to support life—and also why they die so quickly from complicating affections such as pneumonia, which, quickly compromising the little normal tissue that remains, leaves them devoid of excretory function.

(*B*) The *glomerular tissue*. In many, perhaps most, cases of nephritis the glomerular changes are inconspicuous. Careful thought ought to be bestowed upon this fact as bearing upon the treatment of the case. If the glomerular tissue is without important changes, why does the urine diminish in quantity, especially as the blood-pressure is usually increased? And, this being the case, what advantage can it be to the patient to have the blood-pressure further increased by the administration of drugs? Are our conceptions of the dynamics of the kidney correct? Instead of the watery constituents of the urine being transfused through the glomerular tuft in obedience to the variations of the blood-pressure, do we not see in this fact the certainty that the process is after all one of secretion, carried on by the glomerular cells, which are obedient to the laws of transfusion only when in the state of health? It seems to me that this conception is the only one that explains the paradox of hydremia with increased blood-pressure being associated with diminished excretion of water.

In scarlatina and a few other conditions, mostly infectious in nature, the glomerules suffer serious damage, partly from intoxication and partly from the colonization of microörganisms in the capillaries and in the glomerular spaces. Under these circumstances the epithelial and endothelial cells degenerate, round cells infiltrate the tuft, and the partly destroyed glomerule becomes surrounded by a mass of exudate that fills the glomerular space and sometimes extends through its neck a short distance into the tubules. This constitutes the *glomerular nephritis*. All the glomerules affected are thrown out of service, many of them are injured beyond recovery, and the condition commonly proves rapidly fatal, though the fatality is no doubt dependent upon the number of glomerules so affected. There is little doubt that many cases in which some glomerules have been affected get well.

(*C*) The *tubular tissue*. The true parenchyma of the kidney, the uriniferous tubules, is most prone to morbid change. It seems doubtful whether any one dies of

disease without some kind of toxic disturbance of the tubular tissue of the kidney. Except in a few cases of sudden death from accidental injury I feel that I have never seen normal human uriniferous tubules.

The changes pass through the whole gamut of cellular degeneration, from the simple "cloudy swelling" to complete fatty metamorphosis and disintegration. Sometimes the tubules show but a fairly uniform cloudy swelling; sometimes there is added to the almost uniform cloudy swelling irregularly distributed areas of more complete destruction; sometimes there is fairly uniform fatty change. Some cases are peculiar in a marked detachment of the cells and merit the name *desquamative nephritis.*

During the progress of these cellular changes some morbid product of secretion is poured out into the tubules, where it forms the so-called *urinary cylinders* or *tube casts.* The true nature of this substance has not been determined, but it is an albuminoid, varying somewhat in composition in different cases. Various forms have been described, and their appearances are familiar to all who have turned over the well-illustrated pages of modern textbooks upon diagnosis. Cohnheim thought the tube casts to be important factors in the production of the anuria of nephritis, believing that they obstructed the tubules and prevented the urine from escaping, but this view seems untenable, both because we never find distention behind them indicating that they cause obstruction, and because we do find them in large numbers in the urine. They are more probably indications that trouble exists than themselves the cause of any trouble.

The tubular lesions, though not strictly confined to any part of the uriniferous tubule, are most evident in those portions of it that are most active in secretion—that is, the convoluted tubules. The urinary cylinders are most numerous in the straight and collecting tubules, the desquamation most marked in the convoluted tubules.

A Study of the Symptomatology and the Treatment of Acute and Chronic Nephritis.

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We have long known that albumin does not always mean nephritis—it may be functional, prostatic, or even due to spermatorrhea; while nephritis, at least temporarily, without albumin or casts may exist. Casts have also been found in non-inflammatory diseases of the kidney, and hence alone are no certain sign. Clinically, we still divide true nephritis into acute parenchymatous nephritis, chronic parenchymatous nephritis, and chronic interstitial nephritis, and only these three types will be considered, in regard to symptomatology and treatment.

Among sixty cases of nephritis studied, ten only were cases of acute parenchymatous nephritis. They were unassociated with acute infections of any kind, and therefore true types of this disease. Three gave a history of exposure; one of acute alcoholism; two were pregnant; three were convalescent from scarlet fever, four, five, and seven weeks respectively; one gave no history of importance; all were severe cases, and the mortality was 40 per cent. Nine patients were under twenty-seven years of age; six complained of headache; seven presented some edema; six showed pallor (anemia not being determined by blood examinations); eight had a temperature of 100° F. or more, while the other two had each a slight rise above normal; two complained of pain in the back; two showed eye symptoms; and one suffered from uremic convulsions. The urine was reduced markedly in eight cases; the specific gravity was between 1012 and 1030; albumin was present in marked amounts in all cases; casts (principally granular and blood) were persistently present in all; free blood was found in all but one, and it was doubtful.

This brief study of a few cases is enough to impress one that in acute parenchyma-

tous nephritis the urinary study plays the more important rôle, and this is consistent with the opinion of most clinicians. Indeed, this disease is frequently only diagnosed after a careful urinary analysis. The best example of this is seen when acute nephritis develops in the course of an acute infective disease, where no symptoms are present to suggest it, other than the fact that the patient is not progressing well; hence the necessity for careful measurements and frequent examinations of the urine in acute infections, both surgical and medical. The usual symptoms and signs commonly indicative of this disease are emphasized in the cases studied: a history of previous infection, alcoholism, exposure, and pregnancy; fever of 100° F. or more; headache, nausea, and vomiting; possibly dull distress in the back, although this symptom, looked upon commonly by our patients as a sure indication of some trouble in the kidney, is usually in the lumbar muscles and has nothing to do with the kidneys; pallor, or rather anemia, dry skin, loss of strength and even flesh; some edema; and lastly, the urinary findings. Careful urinary analyses are, I think, more important in acute parenchymatous nephritis than in the other two varieties of nephritis. The picture of reduced urine, high specific gravity, persistent albumin of plus amount, casts of epithelium and blood, and particularly free blood, is consistent and impressive. In acute parenchymatous nephritis there is persistent albumin and casts of the well-known types, with dropsy if glomerulous, and with no dropsy if tubular.

Dr. Cabot has emphasized the importance of the amount of the urine in twenty-four hours, the specific gravity and the color, and while probably these are the most important elements, the amount of albumin from time to time, the persistence of albumin and casts, and the presence of blood are of some importance and deserve consideration, especially in this form of nephritis. This is worth mentioning in order that young practitioners and not too-zealous older ones may not be misled by the simplicity of urinary analysis as it is understood to-day, but that they may appreciate the value of urinary findings in the recognition and management of the various forms of nephritis. On the other hand, it is just to say with Meltzer that erroneous results are often obtained from too fine methods of urine examinations. The fault is not in making careful urinary analyses, but in the interpretation of and the significance applied to such findings. Renal infarcts, commonly seen at autopsies, may simulate acute nephritis, but are characterized by sudden appearance of albumin and blood, without dropsy, and sudden disappearance of the same elements. Febrile albuminuria frequently occurring in typhoid fever, pneumonia, and other acute infections can be distinguished by the quantity and persistence of albumin and casts, and the absence of blood and edema.

Acute exacerbations of a chronic nephritis are recognized by the history and by established circulatory signs rather than by urine studies. In acute nephritis the amount of albumin from day to day seems to be a fair index of the successful management; this is not always true in chronic nephritis, where we have to reckon with circulatory changes, which are the best guide.

The diagnosis of acute parenchymatous nephritis rests on, first, the urinary findings; secondly, edema with fever and its associated phenomena; and thirdly, pallor and headache; but all signs and symptoms may be absent, as has been intimated, and the urine alone be the index of the disease. Sudden edema of the larynx or lungs is strikingly for nephritis, and often the acute form, while cardiac dilatations from degenerative myocarditis and uremia are to be feared, after a diagnosis has been made. Acute parenchymatous nephritis is said to be uncommon in typhoid fever, and this is my clinical experience, yet it is surprising, in going over our hospital records, to find persistent albumin and casts, in cases of typhoid fever which have died, diagnosed

toxemia; while as a possible etiological factor in chronic parenchymatous nephritis, former severe typhoid fever is frequently elicited in history taking.

Chronic parenchymatous nephritis is the commonest type of nephritis coming under our care in hospital work. Patients usually apply because of edema, which makes them more suitable hospital cases than those suffering from the vague symptoms of chronic interstitial nephritis. Of this form of chronic nephritis a study was made of forty cases; all but six were between twenty-five and fifty years of age; eight only were women; sixteen gave a history of previous infectious disease; six of chronic alcoholism; three only of syphilis; one of exposure; and from fourteen no history bearing on the present illness could be obtained. Of symptoms, seven complained of chronic headache; all but five had edema, persistent and general; weakness was complained of by all; fourteen showed marked pallor (probably true anemia, but not verified by blood examinations); fever and pain were absent in practically all; and twelve, or 30 per cent, died, while twenty-eight, or 70 per cent, were discharged improved—that is, free from edema and in fair health. Of the urinary findings, the amount of urine was normal or slightly reduced generally; in two cases it was increased; all but one showed albumin in plus quantities and persistent; all but five had granular and hyaline casts upon repeated examinations; the specific gravity was between 1012 and 1025 in all but eight cases; and only one had uremic convulsions. There were no eye symptoms complained of by any patients, and the few eye-grounds examined were free from albuminuric retinitis. Unfortunately no blood-pressure examinations were made in any of these cases.

From a review of these forty cases one is confirmed in his belief that very few, but positive, symptoms and signs are present in this type of the disease, and the urine examination only helps to confirm a diagnosis which is usually suggested on inspection of the patient alone. In other words, the dropsy is so striking as to leave little doubt of the trouble. As one clinician has put it, chronic dropsy with scanty or normal urine of usual specific gravity full of albumin and casts means chronic parenchymatous nephritis. We might add, with accentuated second aortic sound and increased blood-pressure of slight degree, no case should be misunderstood. Pathologically this is the large white kidney of chronic parenchymatous nephritis.

A word must be said about the symptoms of the small white kidney. This condition occurs usually about the age of thirty-five or over, and is marked by loss of flesh and strength, anemia, headache, faulty vision (retinitis), increased amount of urine with plus albumin, but with no dropsy, and presenting vascular changes and hypertrophy of the heart, with high mortality.

Although only three of my cases gave a history of and showed signs of old syphilis, chronic parenchymatous nephritis is believed to be a common result of this disease, some going so far as to say that exposure is doubtful as a cause, and when not due to scarlet fever, alcohol, or lead, it is usually due to syphilis, the nephritis developing within one or two years after infection. It seems rational to believe that probably toxic agents of a chronic nature are always present in producing this disease, and syphilis as a cause may be of more importance than we have heretofore believed.

In regard to urinary analyses in this type of disease, they seem of less importance than in acute parenchymatous nephritis for diagnosis, but nevertheless should be constantly made for confirmatory evidence.

Cases of chronic interstitial nephritis are rarely treated in a hospital—at least where I attend. I have a record of only ten cases in this study. Eight were over forty years of age (although we must not forget that many cases have been reported in young adults); all com-

plained of indefinite gastric or nervous symptoms or of frequent nocturnal micturition; all showed circulatory changes—hard vessels, accentuated aortic second sound, high blood-pressure, and enlarged hearts; and two only presented eye symptoms. The urine in all was increased, of low specific gravity, with usually only traces of albumin and few hyaline casts. No definite history of a cause could be obtained in any case, the trouble developing insidiously. Of four cases the general health was unimpaired, the patients complaining of vertigo and interrupted sleep. Interstitial nephritis is essentially chronic and is not incompatible with long life and usefulness; it is primary, secondary, or arteriosclerotic, the majority of cases being associated with general arteriosclerosis. The signs and symptoms are the most important, the urine, aside from the amount passed, being practically unimportant. As suggested by Bradford recently, there is danger of excluding disease of the kidney from a study of the urine alone in this type, and the profession should be cautioned to examine carefully the cardiac-vascular system and to consider the general nutrition of the patient. All that we need to know in regard to the urine in chronic interstitial nephritis, as an aid to diagnosis, is the amount passed and the specific gravity. But, if further urine studies are made, significance is obtained only by repeated examinations, and even then is of no practical importance.

Some mention should be made here of postural (orthostatic) albuminuria, which is often functional and is characteristic also of convalescence from the parenchymatous types of nephritis, following prolonged local and bodily rest. Postural albuminuria may in some cases be explained by movable (floating) kidney, when no evidence of nephritis is present, but not always. The very insidiousness of this condition makes the symptoms very indefinite, but the signs of resultant damage are more to the point of diagnosis, but necessarily make an early absolute diagnosis almost impossible, and, in most cases, largely presumptive.

Some one has recently called attention to three types of patients suffering from chronic interstitial nephritis: first, those of advanced years suffering from neurasthenic symptoms; secondly, those presenting signs of ill health and wasting; and thirdly, those with striking pallor and demonstrable anemia. The symptoms of this condition are usually in patients over fifty years of age; they complain of indefinite gastric symptoms, vertigo, weakness, nocturnal dypsnea and micturition; suffer from bronchitis, insomnia, cramps in extremities, tingling, impairment of general health, and wasting, with anemia; but the true condition may be recognized accidentally from failing eyesight and headache, or uremic attacks of a mild nature, or from convulsions. The signs are more definite and usually fix the disease. The increased blood-pressure, often over 200 mm. of mercury (and usually the most striking sign), the accentuated aortic second sound, the enlarged heart, the condition of the eye-grounds, with retinal hemorrhages, the sclerosed vessels of the general circulation, and the abundant urine of low specific gravity, are diagnostic. Finally, in the diagnosis of nephritis of the various types, the value of urinary findings is, first (chemically and microscopically), they are of the greatest value in acute parenchymatous nephritis and are more important than the symptoms and signs; secondly, in chronic parenchymatous nephritis they are only confirmatory to symptoms and signs so striking as to make the recognition of this disease very easy; and thirdly, in chronic interstitial nephritis they are comparatively of little importance.

The *treatment* of all forms of nephritis consists of functional rest, or bodily rest, or both. We have no medicines except to meet symptoms and correct complications, and so these cases are handled best by careful management and diet, the use of a few drugs, and occasionally by surgery.

The phenomena of postural albuminuria give us an index to treatment—that is, bodily rest and local kidney rest. Acute parenchymatous nephritis is managed by long rest in bed, from three to six weeks. Once or twice daily a warm sponge bath should be given. Marvel suggests, for a patient in fair condition, a bath of from 94° to 98° F. with passive motion and friction for from six to eight minutes, after which the patient is wrapped in a blanket and remains in it for thirty minutes; and for feeble patients a pack of from 100° to 105° F. for five minutes, followed by twenty minutes' rest. The amount of liquid per day should be about ninety ounces, as suggested by Von Noorden, and about half that should be water, in my opinion. It is my own practice to give normal saline solution by the mouth, from five to six ounces every three hours, in typhoid fever, pneumonia, and all toxic conditions, and when no edema is present, also in acute nephritis, and despite the objection to sodium chloride in this disease, I have seen only good follow. I appreciate the value of a salt-free diet in edema, and observe care when edema is present in all forms of nephritis. Milk, milk and Vichy, buttermilk, vegetable broth, and an increasing non-nitrogenous diet are suitable. Of medicines, when indicated, I use fresh infusion of digitalis, or liquor ammonii acetatis with a small dose of the alkaloid caffeine (gr. j), being sure to have the last dose of caffeine given not later than four or five o'clock in the afternoon, so as not to interfere with the patient's sleep at night.

The chronic forms of nephritis are diseases in which it is particularly fortunate to be rich. They are usually practically incurable, and we can hope only to establish kidney compensation. In chronic parenchymatous nephritis when edema is present, rest in bed; baths and sweats, from eight to ten minutes, once or twice daily; liquid and salt-free non-nitrogenous diet, although if the general nutrition is suffering our diet should be cautiously more liberal, and gradually increased in amount; water actually measured—that is, about forty-five ounces per day besides other liquids; saline purgatives, and the old Niemeyer pill or caffeine (alkaloid). When kidney compensation seems established and edema has disappeared, the urine normal in amount, and health fair, careful living in a dry, warm climate where the altitude is not great should be the portion of the well-to-do. The patient should bathe every day, wear wool or flannel next the skin, get plenty of sleep, drink about forty ounces of plain or some mineral water per day, have only light employment or exercise, be free from care, have a free stool daily, and measure his urine occasionally. His diet should be definitely prescribed, and better largely a salt-free one. It may be said that the salt output should be studied and the salt restricted rather than cut off, as the animal economy seems to need salt for its proper nutrition. The recent studies of a salt-free diet (the only treatment besides rest in many cases), when edema is present, are interesting and instructive, and I think worthy of consideration. The only drug of use in this stage is iron, and as Tyson suggests, only for the anemia when present. Chronic interstitial nephritis is also a disease for modified rest treatment. The patients are suffering from the damage to the circulation and heart, and should be directed to follow a quiet life, free from care, and guided along the same lines as those suffering from chronic parenchymatous nephritis. One aim should be to reduce blood-pressure, never forgetting that a certain increase above normal is consistent with advanced years and should not be greatly reduced. Of drugs, nitroglycerin, iodide of potassium, or better iodide of soda, in small doses and over a long period, should be tried.

Only one word in regard to uremic convulsons: I cannot agree with my old teacher in this matter, for I give morphine at once when the convulsions occur, irrespective of the type of the disease. I have

also given chloral hypodermically with happy results in uremic convulsions. I have had no experience with surgery as applied to these types of kidney diseases, but after reading Ramon Guiteras's statistics collected from forty-two surgeons where the mortality of operation is 25 to 26 per cent, and only 17 per cent of cures in 160 cases, I am inclined to stick only to medical management.

The Treatment of Nephritis.

By James Tyson, M.D., Philadelphia,
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The object of the science and art to which we are devoting our lives is the prevention and cure of disease. Not so very many years ago cure claimed most attention, was most sought, while prevention played a minor rôle. At the present day while we abate in no way our search for cure, prevention has come to the front and has of late probably been far more successful in checking the ravages of disease than cure.

Bright's disease is no exception to the rule, and I believe I can be of more use to you by dwelling first on measures of prevention. Although we have taken great trouble in the past to distinguish parenchymatous and interstitial nephritis, in point of fact all nephritis, except a few malignant cases of the diffuse form and suppurative nephritis, or surgical kidney, is parenchymatous, by which I mean the process begins in the living cells of the tubules and causes their ultimate destruction and disappearance, to be followed by a substitution of connective tissue. The chief difference lies in the fact that in the so-called parenchymatous nephritis the cell changes are more rapid, though less complete than in the interstitial form, and associated with a primary proliferation and increase of cells, resulting in a pathological product very different in appearance and size, at least in typical cases. In both cases the immediate cause is an irritant acting on the renal cells. The object of the preventive treatment is to avert the irritant, while the object of the curative treatment may be said to subdue the irritant and remove its remote results.

In order to avert the irritant we must know what it is. Its varieties are many and unfortunately are often quite undiscoverable. The best known of them are the causes of the infectious diseases such as scarlet fever, diphtheria, yellow fever, cholera, measles, typhoid fever, rheumatism, and even tonsillitis. Others are overeating and drinking, and especially an excess of proteid ingesta and alcohol, and to a less degree of tea and coffee. To these must be added toxins of the nature of ptomaines generated in intestinal putrefaction and fermentation, all of which throw an excess of work on the kidney cells, resulting in their destruction. The result is imperfect elimination and consequent accumulation of toxic substances, and as the result of these, degenerative changes in the vascular system and in uremia in its various manifestations. As we prevent infectious diseases from occurring and are prompt in their successful treatment, we therefore eliminate nephritis. This covers a large number of cases.

After these come alcoholic excesses and excessive eating, also the ingestion of strong tea and coffee in large quantities. The last occurs more frequently among the lower classes than is commonly supposed. It is not uncommon for the servants of well-to-do families to have tea twice a day, at 11 A.M. and 4 P.M., in addition to the tea and coffee they may take in the morning and evening. By watching such cases as these, therefore, to which almost no attention has heretofore been directed, we may further cut out cases of nephritis, more particularly the chronic forms.

Moreover, there is no doubt that exposure to cold and moisture, whatever its mode of action, whether as a direct cause or favoring the operation of other causes mentioned, is responsible for a number of cases of acute nephritis, as when a man while overheated and perspiring becomes suddenly drenched, or, lying down on the damp ground, falls asleep and acquires an acute nephritis forty-eight hours later.

So much for the preventive treatment; now as to the curative. The further management of nephritis is more easily considered by separating the acute from the chronic form. The treatment of the former is far simpler, followed by prompter results, and more easily disposed of—in fact, in a limited discussion need claim but a few minutes. Absolute quiet, a milk diet of rather poor than rich milk, purgatives, diaphoretics, and diluent diuretics are the measures usually efficient in the average case. For complications, including uremia and dropsy, the same measures judiciously increased are also applicable. I am still timid of the use of morphine for the control of convulsions, although less so in the convulsions of so-called parenchymatous nephritis as contrasted with interstitial inflammation in which the danger is greater, while the difficulty in diagnosis in certain cases makes it no easy matter to decide whether the drug is safe or not. On the other hand, chloroform inhalation to control convulsions, a few drops being placed on a handkerchief for inhalation, is an efficient and safe remedy for this purpose. The effect may be kept up by chloral enemas. As much to be recommended is venesection, followed by hypodermoclysis or intravenous injection of normal salt solution. The latter without venesection may be dangerous, and I pause to emphasize this statement: I think there is danger of overcharging the venous system and precipitating the death of the patient. Nor is it always well to push treatment too rapidly in uremic coma and convulsions. The condition is such an alarming one that we are inclined to try rapidly one remedy after another, when it would have been wiser to make haste more slowly and give nature a chance to assert herself.

A nicer judgment is often required to treat chronic cases than acute. In the first place there are quite a good many chronic cases better without any treatment except a simple hygienic and dietetic course, and this of a not too active kind. These are generally cases which have been discovered by the life insurance examiner, and were previously unsuspected. It is a great mistake to treat such cases actively. I usually say to such patient that it is fortunate the albuminuria was discovered in order that he may intelligently avoid the causes which may make him worse, and that, such precautions being taken, he is no worse off than if he had no albuminuria. Practically the same injunction may be given to similar instances discovered by the physician in a systematic study of a case. Here the advice should be to avoid exposure, fatigue, and overwork, to retire early and seek abundance of rest, along with a minimum but not exclusion of proteid food. Especial stress should be laid on moderate eating of all kinds of food and the exclusion of alcoholic drinks, while the free ingestion of liquids between meals should be encouraged, although this, too, may be overdone. Happily the red meat craze is dying out. I never did join in it. Red meats contain no more nitrogen extractives than white. Boiled meats are supposed to be slightly less injurious than roast, as in boiling some of the extractives are dissolved; but it is a matter of quantity rather than quality.

Warm bathing, especially at bedtime, followed by friction is a useful hygienic measure. It should be daily when possible. Cold sea bathing, especially long submersion, is harmful.

In the matter of medical treatment directed to the kidney, I know of no drug which will create new renal cells to substitute those replaced by connective tissue, but conditions favoring nature's own efforts in this direction are those already mentioned. Our object must be to save the kidney from irritation. There is perhaps even in the mild degrees a tendency to anemia which requires the administration of iron, but this remedy in the shape of the well-known Basham's mixture is too carelessly administered and often does more harm than good by causing constipation and locking up other secretions. I do not give it as much as I used to. Iron should be given in small doses, and I often give the tincture of the chloride in combination with very small doses of bichloride of mercury freely diluted. The bichloride checks abnormal fermentations and the generation of toxic

substances in the stomach and intestines. A regular, sufficient action of the bowels tends to the same end. Purgatives acting on the liver are especially serviceable in ridding the blood of toxic substances. To this end a weekly or biweekly dose of calomel is suitable. The free action of the skin diminishes the demand on the kidney. The iodides dilate the blood-vessels and facilitate the onward movement of the blood, hence some physicians hold that the more or less constant administration of the iodide of sodium or potassium is necessary in every case of chronic nephritis. They are also a good diuretic in most cases. They may have some tendency to remove fibroid overgrowth. I use them a great deal. It is to the next stage, however, in which there is the constant presence of an appreciable quantity of albumin and numerous casts, to which these last remarks apply. You may want to use one of the newer diuretics, of which aceto-theocin is a good sample. It is one of the caffeine group, of which diuretin is also another. They are said to dilate the blood-vessels of the kidney as well as to increase the strength of cardiac contraction. The dose of theocin is three to five grains every six hours. It is a more reliable drug than diuretin, although I have known the latter to act very well, and one must have many strings to one's bow.

The complications of chronic nephritis include the dropsy and uremia of the acute form and call for much the same measures. They are, however, more difficult to relieve and ultimately carry off our patient.

In uremia venesection with hypodermoclysis, purging, and sweating are the sheet-anchors. Another valuable measure is the injection high up in the colon of hot salt solution, three or four times a day, starting with the solution at 110° F. Most efforts in this direction are failures because not correctly carried out. The hips should be elevated, a rectal tube passed high up in the rectum, and the water allowed to run in very slowly. It has even been suggested to have the fluid enter almost drop by drop. In this way large quantities may be introduced, but the temperature of the water necessarily falls. If there is not much edema, some of the hot fluid may be left in the colon; if there is much edema, the limbs may be scarified or Southey's tubes inserted and the water thus run off. Nitroglycerin is a remedy often very carelessly used. It is supposed to have a diuretic effect; I confess I have never seen any reason to believe it has any. On the other hand it is harmless and tends to relieve any high tension that may be present.

Very different is it when we have actually to deal with the high tension of granular kidney. A slightly higher arterial tension than is physiological is necessary to a proper function by a diseased kidney; but high degrees, say a pressure exceeding 160 milligrammes of mercury, are harmful. Here nitroglycerin is of signal service. One hundredth of a grain may be given as often as every two hours until the desired effect is produced. A favorite combination of Sir Lauder Brunton is potassium bicarbonate gr. xxv, potassium nitrate gr. xv, sodium nitrite ½ gr. In the opposite condition of low tension digitalis and caffeine must be substituted, and the sphygmomanometer may be necessary to determine which is present.

Cardiac failure must be added to these complications, increasing and aggravating both dropsy and uremia and being itself sometimes a cause of sudden death. It succeeds upon the hypertrophy so frequently associated with chronic Bright's disease, and is commonly the direct result of dilatation of the heart. Strychnine and digitalis or strophanthus are among the best remedies. In this condition the circulation must not be overloaded with water. The use of water must rather be restricted.

You will want to know what I think of Edebohls's operation of decapsulation for the cure of chronic Bright's disease. I believe the operation has a place in the treatment, but I would not call it curative. Like so many treatments it has disappointed expectation, and probably in consequence is too much ignored. I have thought the better class of surgeons have not given this operation enough consideration, prob-

ably, too, because the conditions supposed to indicate it are so purely in the province of the physician. It is an operation whose suggestion must come almost wholly from the physician, and he is sometimes not as ready to suggest operation as he ought to be. I have seen life saved and prolonged by it, and I am sure this may happen again and again. The cases in which it should be done are generally those of parenchymatous nephritis with extensive anasarca, which refuse to yield to other treatment.

In interstitial nephritis the operation is less hopeful, as there remains less of the parenchyma of the organ to resume its function. There can scarcely be a doubt that the relief comes through diminished tension, illustrated by a case of my own, a typical case of chronic parenchymatous nephritis, in which the patient complained especially of a dull pain in the region of the left kidney along with a sense of aching fulness or distention in the same vicinity—in the patient's language, "a feeling as though there was an abscess there." In the fall of 1906 the kidney was exposed, its capsule stripped, and the organ replaced. In twelve days the patient was taken from the hospital, and a journey of 140 miles was made with very little help on and off the trains. Previous to this there was large albuminuria, dark granular and oil casts. Succeeding the operation he gradually improved, and with the approach of spring began to take advantage of the fair weather to get out-of-doors, then gradually to resume business, until recently he wrote that he feels in as good health as he ever did, and outside of not being as strong as he would like to be, he has no complaints.

With what may be called the organic treatment of nephritis I have had no experience. Indeed, to suppose that nephritis can be cured by feeding its subject with fresh kidneys seems to be so absurd that the idea is repugnant to me. Still I may be wrong. Dr. Oliver T. Osborne in a paper on "Kidney Insufficiency and the Treatment of Uremia"[1] says he certainly has seen "life prolonged in nephritis by feeding fresh kidneys," though he has not had much result from nephritin tablets. He says also that "thyroid extract certainly aids in preventing convulsions in uremia and is," he believes, "the most efficient preventive of that symptom." I have never used it.

The saddest picture in the drama of Bright's disease is that of albuminuric retinitis. It is certainly in the majority of cases the beginning of the end, and the shadow of darkness grows irresistibly until the valley is crossed. Can anything be done to avert it or to diminish it? To avert it there is nothing but what has been laid down in my previous remarks. To diminish it we must at least make an effort by mercurials and the iodides to arrest the process. Patients thus afflicted should be kept on the most stringent diet, and continuously on such doses of the biniodide of mercury, or, better, the iodide of sodium with the bichloride of mercury, as can easily be borne, and all strain upon the eyes should be guarded against. The progress of the disease varies, extending more rapidly in some cases than in others, but I sometimes think the prognosis of the ophthalmologist, who is so often the first to make the discovery, is needlessly grave. One thing I have learned rather late in life which is of extreme importance, and which I would strongly urge upon you, is to insist upon an ophthalmoscopic examination in every case of Bright's disease at least twice a year.

Renal headache is one of the most severe and sometimes gravest of symptoms. It occurs in two forms: first, in the shape of a more or less severe dull pain, often in the occipital region, lasting a variable time; and secondly, a short, sharp, shooting pain, which is often the prelude of uremic convulsions. Both are probably due to the causes of uremia, whatever these may be. The treatment is in all cases eliminative: in the first form, that of the uremic convulsions, at the time; in the second, by more deliberate measures. A remedy of this class very highly recommended by Dr. Samuel West, and with which I have had

[1] *Journal of the American Medical Association*, Aug. 24, 1907.

some experience, is pilocarpine nitrate or muriate, of which the beginning dose is one-twelfth grain rapidly increased to be sufficient to produce an effect on the skin. Dr. West regards it as an efficient remedy in uremia. These headaches are sometimes so severe as to require hypodermic injections of morphine to relieve them; but before this is done, caffeine, nervous sedatives, and counter-irritation should be tried.

The Circulation in Nephritis.

By William B. Stanton, M.D.,
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The close relationship between disease of the kidneys and enlargement of the heart was first pointed out by Richard Bright. Subsequent investigation has confirmed his findings and given rise to many theories which attempt to explain this connection. In order to appreciate the difficulties of the problem, it may be permitted to briefly review what is known regarding the anatomy and physiology of the kidney.

The sectioned kidney surface shows a cortex comprising an outer third which contains the secreting part of the organ—the Malpighian bodies and the convoluted portions of the urinary tubules. The medulla, comprising the inner two-thirds, contains the collecting tubules, blood-vessels, and connective tissue. The blood-supply of the cortex is of special interest. The arteries entering the hilum pass up or toward the surface between the tubules, until they reach the junction of the cortex with the medulla, where they bend sharply to form a series of horizontal arches from which are given off two sets of vessels—one set passing to the medulla which it supplies, the other passing into the cortex. These cortical branches are peculiar. As they pass toward the free surface they give off short lateral twigs, which supply the afferent vessel of the Malpighian bodies. This afferent vessel divides into groups or lobules of convoluted capillaries—the glomerulus. These capillaries terminate in the efferent vessel, which is smaller than the afferent vessel, but still conveys arterial blood. The efferent vessel soon breaks up into capillary networks which surround the tubules of the labyrinth. This second set of capillaries is taken up by the interlobular veins which accompany the arteries. The blood passing through the kidneys thus meets with unusual obstruction because of the two sets of capillaries.

The unit of the kidney is the tubule. This begins as a blind extremity, Bowman's capsule, which is invaginated by and completely surrounds the tuft of capillary vessels known as the glomerulus. These two structures constitute the Malpighian body. After the capsule of Bowman the important parts of the urinary tubules are the two sets of convoluted tubules which are lined with cuboidal epithelium.

There are two chief theories as to the physiological process of the urinary secretion. Bowman thought that the glomeruli secreted only water, while the specific urinary elements were furnished by the epithelial cells of the tubules, to be washed onward by the onflowing water. C. Ludwig assumed a diluted urine was secreted in the capsules. In passing through the tubules the excess of water was removed, and so the ordinary consistency was reached. It is evident that the secretion of urine is much more than a simple physical process, and depends upon the vital activity of the cells concerned. The urine is composed of the urinary water and the urinary elements held in solution. According to Landois, the urinary water is secreted chiefly in the capsules, and the amount depends primarily upon the blood-pressure within the renal artery. The blood-pressure, however, is not the only factor, as the vital activity of the cells of the capsule plays an important part. This vital activity is proven by the fact that the cells become paralyzed by temporary occlusion of the renal artery, and for this reason the kidney no longer secretes, even when the circulation is restored. Another proof is the fact that temperature of the urine is often higher than that of the arterial blood, and while the serum-albumin of the blood is retained, egg-albumin when injected into the blood is secreted. The dependence of the urinary secretion upon the blood-pres-

sure is shown by the increase in urine following increase of the total contents of the vessels. Increase of blood-pressure beyond a certain level may even cause albumin to pass into the urine. Loss of water, as in profuse sweating or diarrhea, causes a diminution in the urine output. Increase in the heart's activity with consequent increase of arterial tension and rapidity of the blood current produce increase of urine, while cardiac weakness causes a decreased output. In animals, artificial stimulation of the vagi, with slowing of the heart and reduction of arterial pressure, caused diminution in the output of urine. When the aortic pressure fell to 40 mm. of mercury, all secretion of urine ceased.

While the quantity of urine depends upon the above facts the concentration depends upon the amount of urinary constituents in solution. These substances appear to be taken from the blood by the independent activity of the cells of the convoluted tubules. This independent activity is shown by the fact that indigo carmine, when injected into the blood, can afterward be found in the cells of the convoluted tubules and not in the capsules. The indigo carmine may also be found in the lumen of the tubules further on, where it has been washed by the passage of the urinary water. If the cortex of the kidney is destroyed previous to the injection of the indigo carmine, this substance is found to remain in the cells of the convoluted tubules. This has also been demonstrated in regard to urea, biliary pigments, the iron salts of the vegetable acids, hemoglobin, and milk.

The nerve supply of the kidney is not completely known. So far only the effect of the vasomotor nerves upon the filtration of urine from the renal vessels has been studied. The nerves for each kidney are apparently derived from both halves of the spinal cord. The center for the renal vasomotor fibers is situated in the floor of the fourth ventricle, in front of the origin of the vagus and very close to the vasomotor center of the liver. Injury to renal vasomotor center, or to any part of the active nerve path, causes vasodilatation with increase of urine. The splanchnic nerve also contains some renal vasomotor fibers. If the splanchnic is paralyzed, there will be a temporary increase of urine which soon disappears because so much blood finds its way to the large intestinal areas. Irritation of this nerve causes the opposite effect. The vagus appears to be the vasomotor nerve of the kidney, and is thought to possess trophic functions as well.

A consideration of the facts just presented enables one to appreciate the very great effect that disease of the kidneys must produce upon the circulation, and especially upon the heart. As already stated, the frequent enlargement of the heart was noted by Richard Bright. He believed that there was a change in the constitution of the blood which acted as an irregular and unusual stimulus directly upon the heart, or that it affected the smaller vessels and capillaries in such a way as to require a greater exertion to drive the blood through the terminal vessels of the arterial system. The cases of Bright, like those later published by Traube, showed that the left ventricle was especially affected. The large number of cases that have been published permit of certain definite ideas as to the frequency with which cardiac hypertrophy occurs in the different forms of kidney disease. Enlargement of the heart occurs most frequently in chronic cases, and especially cases of genuine contracted kidney. It is, however, not always present; the percentage, according to Senator, varying between 50 and 90. In the cases of parenchymatous nephritis, enlargement of the heart may be present, especially when contraction follows. Ordinarily it is infrequent. In acute nephritis of certain kinds hypertrophy of the heart is absent, as in nephritis from arsenic and phosphorus, or that arising in the course of typhoid fever and diphtheria. In primary, acute Bright's, which, according to Krehl, is probably infectious in character, and in scarlatinal nephritis, increased blood-pressure and cardiac hypertrophy are nearly constant. Friedlander (*Archiv für Physiologie,* 1881, p. 168) found hypertrophy and dilatation of the heart in children dying with scarlatinal

nephritis amounting to 50 per cent increase in weight. Those dying of the scarlatina without nephritis showed no enlargement. Four adults, between twenty and thirty years of age, showed no cardiac hypertrophy after death from scarlatinal nephritis.

While the enlargement is most noticeable in the left ventricle, more recent observations have shown that in many instances the right ventricle and the auricles were also affected, according to Krehl, in 82 per cent of those examined by the method of Müller. Investigations of the heart made by C. Hirsch after the method of Müller give a good idea of the average findings. By this method the heart is carefully cleaned of fat, and then divided into its anatomical parts, and the auricles and the ventricles weighed separately. These weights are then compared with the total body weight. In his thirteen cases of contracted kidney the left ventricle was exclusively enlarged four times, with an average increase of 43 per cent above the normal. In seven cases the right ventricle showed a hypertrophy, averaging about 18 per cent above the normal size, while in these same cases the hypertrophy of the left ventricle amounted to an average of 56 per cent. In the remaining two cases the increase was 74 and 129 per cent respectively on the right, and 76 and 188 per cent on the left. Of these cases reported by Hirsch, nine were complicated by an existing arteriosclerosis.

This enlargement of the heart presupposes an increase in blood-pressure, and the results of blood-pressure estimations have been in accord with the pathological findings. The results of Buttermann (*Deutsches Archiv f. Klin. Med.*, vol. 74, 1902) may be taken as a fair example of the usual findings. In eighteen cases of acute nephritis, the elevation of pressure was found to come on quickly—in one case there being an increase of 50 mm. within forty-eight hours after the beginning of the albuminuria. With improvement the pressure returns toward the normal. At the time of the increase in blood-pressure the second sounds of the heart at the base were accented, and there was a heaving apex beat. Occasionally there was dilatation of the heart cavity with or without a muscular insufficiency of the mitral. This dilatation can occur in combination with increased blood-pressure, and according to Buttermann is an evidence that the heart is able to meet the increased demands only in part. In thirteen cases of contracted kidneys the pressure was constantly elevated. In four cases of amyloid kidney the pressure was normal. In thirteen cases of chronic parenchymatous nephritis the pressure was variable—in one group, subnormal; in a second group, normal; in a third group, on the upper bounds of the normal.

The facts in the matter are so well established that a collection of blood-pressure estimations is of little interest. The important question is why does the heart hypertrophy? or what amounts to the same thing, why should the blood-pressure be increased? The theory of Bright has already been mentioned. Other theories have been advanced. Traube thought the obliteration of the numerous blood-vessels in the kidney or their compression by disease products offered resistance to the onflow of blood, while there was also a retention of water in the system which gave the heart more liquid to move in a given time. This theory is defective, because removal of the kidney or ligation of the renal artery has no permanent effect upon the aortic pressure. Also it has been found impossible to raise the normal arterial pressure by injecting salt solution in the veins of animals. Johnson thought the retained material in the blood stimulated the smaller arteries to contraction through the vasomotor system, and thus secondarily increased the work of the heart. He believed that this led to hypertrophy of both the heart and the muscular wall of the vessels. The objection to this is that hypertrophy of the muscular layer of the vessels is not always found. Gull and Sutton thought the kidney disease was merely a concomitant of "arteriocapillary fibrosis" which was the real cause of the hypertrophied heart. Da Costa reported changes in the nerve cells of the renal ganglia and in the lower ganglia of the

cervical sympathetic. The renal disease as well as the cardiac hypertrophy was attributed to this nerve disease. This observation has never been confirmed. For a time it was thought that through the disease of the kidneys a change was produced in the blood by which its viscosity was increased—that is, there was an increase in internal friction, thus augmenting the work of the heart. The researches of Hirsch and Beck (*Deut. Archiv für Klin. Med.*, vols. 69-72) upon the viscosity of the blood in nephritis were unable to demonstrate any increase.

The most tenable theory at the present time is that of Bright, and the majority of observers, as Krehl and Senator, are inclined to believe that the cause of the increased pressure lies in some disturbance or irritation of the walls of the smaller vessels. This alone cannot explain the hypertrophy of the right side of the heart, and Senator believes that the retained excrementitious products may act upon this as a direct irritant.

The facts at our command, then, are that in many instances of kidney disease the systemic blood-pressure is elevated, and the heart hypertrophied. While we are not in a position to measure the pressure in the pulmonary circulation, it is reasonable to suppose that it too is increased, because in carefully examined hearts the right side of the heart is also hypertrophied. This hypertrophy is not due to a failure of the left heart, thus throwing additional work on the right side, because the right-sided hypertrophy is found in hearts with perfectly competent valves, and because the enlargement of the heart is predominantly a true hypertrophy with little or no dilatation.

The theories which have been offered to explain the cardiac results of Bright's disease are incomplete, because the attempts have been made along purely physical and mechanical lines. We have to do with exceedingly complex structures performing different kinds of work in different portions. From the researches already quoted, it is seen that the Malpighian bodies and the convoluted tubules may and do work independently of each other, and presumably either of these may be diseased, while the other remains, for a time at least, practically normal except for secondary effects. Thus the blood passes first through the glomeruli, and from it the capsule secretes water and certain diffusible salts. It then passes into capillaries which surround the convoluted tubules, and from this same blood these tubules secrete the specific urinary elements. It is not hard to suppose that primary disease of these tubules would produce stagnation or obliteration in their capillaries with consequent back pressure upon the glomeruli. It is in this way that a weak heart with following congestion of the kidneys acts to produce diminution of the urine. Disease of the capsules may also occur independently, and the difference in blood-pressure may be due in part at least to the portion of the kidney diseased. While the kidneys may show the most marked changes at autopsy, we cannot be sure that they were primarily affected, and thus produce the circulatory changes, or whether they were not diseased by their efforts to rid the body of some poison. The poison of gout or from lead may produce typical contracted kidneys.

The general increase of pressure must be caused by vasomotor stimulation, but whether this acts directly upon the vessels' walls as does adrenalin, or through the vasomotor center, is as yet unknown. Whether this pressure becomes actually high and hypertrophy of the heart ensues will depend upon the reserve power of the heart and its nutrition. In conditions of low vitality, as in tuberculosis, increase of blood-pressure and cardiac enlargement rarely occur. In the few cases where this was found, the tuberculosis was arrested.

Much stress is laid upon the clinical diagnosis of cardiac hypertrophy in kidney cases. The classical signs are accentuation of the aortic second sound, heaving apex beat, and a hard pulse. If all these are present typically the diagnosis is certain, but the converse is not true. Accentuation of the aortic second sound is a very frequent occurrence in tuberculosis, where the blood-pressure is universally low. This is

most often found in lesions of the right apex, whether they be old or new. The highest blood-pressure record that I have, 260 mm., was in a case seen for Dr. Hobart Amory Hare, and here all the heart sounds were rather weak and the apex beat not palpable. The only certain sign of increased pressure is a blood-pressure estimation with a reliable apparatus.

Finally, a word may be said in regard to the significance of the heightened pressure. I think we have a right to assume that this is a conservative effort upon the part of nature to carry on the functions of the body. If this is so a marked reduction of the pressure by the means of drugs may produce disastrous results. Correction of mode of life and regulation of diet and exercise will aid in maintaining the heart's nutrition, and thus enable it to keep pace with its work. Until we are more familiar with the nature of the poisons or diseases with which we have to deal in cases of so-called chronic nephritis, we will get better results by sustaining the heart than by trying to lower the pressure.

The Surgery of Movable Kidney.

By Dr. J. Chalmers Da Costa,

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The first obligation of surgery is not to do harm. I am certain that in many cases of movable kidney operation is not indicated. The kidney is not normally a fixed organ; it is naturally mobile. It is only when the mobility is excessive that we are justified in regarding it as abnormal. The normally attached kidney moves with each respiratory excursion. This may be demonstrated in any person in whom the organ is palpable; and the lower pole of the kidney, especially of the right kidney, is palpable in many individuals. The right kidney is placed on a slightly lower level than is the left, owing to the large liver above it. It may be felt in thin subjects with relaxed muscles, in whom the ileocostal space is wide. Physiological mobility is demonstrated clearly during operations that expose the kidney, provided the organ is not anchored by inflammatory adhesions.

Whereas some mobility is natural, extreme mobility is certainly harmful. That the fixation of a normally mobile organ may be injurious is probable; that the fixation of an abnormally mobile kidney is necessary seems to be proved. Our first proposition is that a movable kidney should not be operated on at all, unless we are convinced that the mobility is doing harm or is so great in range that it is certain to do harm. If we are sure that there is excessive mobility, and that it is doing or is sure to do harm, and if conservative methods have failed or are obviously useless, we operate. In doing so, we choose the uncertain and lesser evils of fixation instead of the certain and progressive dangers of extreme and increasing mobility.

It is, therefore, evident that in a case of movable kidney a surgeon must, first of all, determine accurately that slight, marked, or great mobility really exists, and whether or not mobility is doing or is certain to do harm. Dr. Harris, of Chicago, has wisely suggested that we should describe three degrees of mobility. The terms that he has introduced are very useful clinically, and I always employ them. The *first degree,* or *slight mobility,* includes those cases in which, perhaps, a considerable extent of the kidney can be palpated, but in which it is not possible to bring both hands together above the organ, when one hand is held in front and the other is held behind. The great majority of cases seen in practice are instances of the first degree of mobility; and in most of these cases I believe that conservative treatment is indicated. The *second degree,* or *marked mobility,* includes those cases in which the hands can be brought together above the organ; and all these cases require operation. The *third degree,* or *great mobility,* includes those cases in which the kidney drops to the pelvic brim or, perhaps, moves to beyond the umbilicus. All these cases require operation.

In examining a patient for movable kidney, I employ the method of Kendal

Franks. The patient is placed recumbent. When the right kidney is being examined, the surgeon stands at the patient's right side, places the four fingers of his left hand in the loin below the twelfth rib, and holds the thumb, with light pressure, in front and just below the ribs. The patient is told to take a deep breath, to hold it a moment, and then, on receiving the command, to empty his lungs slowly. Just before he begins to expire, the surgeon makes an effort to approach his thumb toward his fingers, the thumb being thus pushed up toward the ribs; and as the patient expires the digit follows the rising liver. Even a slightly movable kidney can thus be palpated. If the kidney is movable in the second or the third degree, the organ at the termination of expiration will be caught below the surgeon's thumb and fingers. When this occurs, if the grasp is slightly relaxed, and if the right hand is used to press upon the kidney, that organ will rapidly slip upward between the thumb and the fingers of the left hand, feeling like an object that has been greased.

If the left kidney is to be examined, the surgeon stands at the left side and employs the right hand to make the diagnostic grasp. Instead of using the fingers and thumb of one hand in examining for movable kidney, the surgeon may prefer to place the fingers of one hand in the loin and the fingers of the other in front and below the ribs, giving the patient the same directions as to filling his chest and then emptying it.

Having determined that there is mobility beyond physiological limits, the next question is, How shall we treat the case? In movability of the first degree, without local or general symptoms, there being merely the evidences of hysteria or of a neurotic state, operation is contraindicated. In such cases, anchoring the kidney will not cure the neurotic state. In many of the victims of movable kidney there is a highly neurotic condition, general prolapse of the abdominal viscera, and bilateral renal mobility, the mobility being worse as a rule on the right side than on the left. In such a case, even if both kidneys are operated upon, the general splanchnoptosis will not be cured and the neurotic condition will not be amended.

If, in a case of mobility of the first degree, distinct local symptoms exist and persist, harm to the kidney is threatened or has begun; and if conservative treatment fails, operation is indicated. If the range of mobility is found to be gradually, but certainly, increasing, in spite of non-operative treatment, operation is indicated in order to prevent an otherwise inevitable outcome—that is, the attainment of marked or of great mobility. The deduction from this observation is obvious: it is that a patient with movable kidney of the first degree should be examined at regular intervals, in order that the surgeon may determine whether the range of mobility is increasing.

I have used the term "local symptoms," and it is proper to state what is meant by it. Three local symptoms are of great importance, viz., severe renal pain, distinct renal tenderness, and enlargement of the kidney. Enlargement means the beginning of hydronephrosis. Palpation detects enlargement only when it is considerable. The beginning of dilatation can be detected only by catheterizing and injecting the ureters, and thus finding that the renal pelvis of the movable kidney holds an inordinate amount of fluid. In every case of distinctly movable kidney the ureter should be catheterized. This point has been insisted upon by Dr. Kelly, of the Johns Hopkins Hospital. If catheterization indicates that dilatation has begun, operation is indicated. In every case with marked mobility and in every case with great mobility the operation is indicated, whether there are local symptoms or not, and whether there is dilatation or not. In such cases a let-alone policy will be followed, sooner or later, by trouble. The kidney will inevitably come to grief and undergo structural changes.

In cases in which the kidney is freely movable, a Dietl's crisis may occur at any time. A Dietl's crisis is an attack of pain in the epigastrium, in the loin, or

in both regions. The pain frequently radiates, as does the pain of nephritic colic. There are also nausea and vomiting; tenderness; weakness, or even collapse; not unusually abdominal rigidity; and sometimes nervous chills. Elevation of temperature is frequently noted after such chills. A Dietl's crisis is due to twisting of the renal pedicle, which is productive, as Dr. Charles P. Noble has said, of congestion or hydronephrosis, or of both. There is great variability in the violence and danger of these crises. A slight and temporary twist of the pedicle may simply cause hematuria, albuminuria or casts, renal pain, abdominal pain, or dragging in the loin. A more severe and protracted twist may produce serious hydronephrosis, or even complete obstruction of the renal vessels and ureter. I operated upon such a case during its active stage, and found the kidney in a condition bordering on necrosis. If a definite and positive crisis occurs, others are certain to follow, and operation is positively indicated.

In cases of movable right kidney, I am often in doubt whether a pain in the right iliac region is of renal or of appendiceal origin. Some surgeons would easily and comfortably settle the matter by saying that catarrhal appendicitis is particularly liable to arise in persons with movable kidney, and that the pain of such a patient is appendix pain and calls for the removal of the appendix. I have never been convinced that appendicitis is associated with movable kidney with any particular frequency. In fact, Dr. Howard Kelly states specifically that the observations made at Johns Hopkins Hospital indicate that it is not even very commonly associated with movable kidney. Kelly shows how one may determine whether this pain is renal or appendiceal. He injects the ureter and overdistends the renal pelvis. If such distention produces the characteristic pain in the right iliac region, he concludes that the pain is renal. If it fails to do this, the pain is appendiceal.

If it is decided that the pain is appendiceal, one should remove the appendix, as well as anchor the kidney. The appendix may be removed through a second incision, or both operations may be done through the one incision in the back. I have done this several times, with ease and satisfaction.

Suppose that it is decided to employ conservative treatment, how shall it be carried out? Prolonged confinement to bed, with forced feeding, leads to a deposition of fat, and in some cases to the fixation of the kidney. This form of treatment, however, is possible only in certain individuals.

Any patient being treated conservatively should be cautioned to refrain rigidly from heavy lifting, sudden and forcible movements, and violent exercise. This care is the basis of all success. It is entirely out of the question for any pad to press the kidney into place and hold it there. Such a pad can only be pushed against the kidney by pushing in the belly-wall. If such powerful pressure is continued, it is sure to be harassing and often becomes unbearably painful. If a patient wears such a pad, each increase in intra-abdominal pressure caused by coughing, lifting, straining, etc., lifts the pad away from the kidney and allows that organ to slip below. Then, as relaxation occurs, the pad sinks in, perhaps gets above the kidney, and may actually hold that organ displaced downward.

It thus becomes evident that kidney-pads may be productive of great harm. A broad abdominal bandage is much better than the pad. It lifts other organs up against the kidney, and may thus help to hold that organ in place. I usually employ the corset devised by Dr. Gallant, of New York City, and find it very satisfactory in many cases. Dr. Gallant has placed on record the proper way to apply and to use this corset. My friends, Drs. Rugh and S. S. Cohen, have been using straps of adhesive plaster with very great satisfaction. They use the old-time moleskin plaster, and apply it in a particular manner, by the use of several

strips. They will report their results in a short time.

When a decision to operate has been reached, the next question is, What sort of operation shall be done? To give success, any operation should restore the kidney to—or at least nearly to—its normal place, and should put it in practically its normal relation to surrounding parts. I have recently discussed in some detail the question of operations for movable kidney, and I shall not repeat those arguments here. Suffice it to say that I do not believe that any operation that places stitches through the kidney substance should be employed. They loosen almost immediately, and they may do harm by inducing urinary fistula. I do not believe in any operation that catches the kidney by its rolled-out capsule, so that the convexity of the kidney is anchored to the structures of the back. This position is an abnormal one, puts a quarter of a twist in the ureter, and may be followed by the very hydronephrosis that it has been practiced to avert. The operation that places stitches in the posterior surface and the lower half of the kidney lifts the organ much more nearly into a normal position than do the other methods of suturing, but has a tendency to tilt the upper pole of the kidney forward, a position that is peculiarly unfavorable and apt to cause trouble to the surrounding structures, especially the bile-ducts.

The operation that seems to me to put the kidney most nearly into its normal position and to give the best assurance of solid fixation is that by means of the gauze pads—the operation usually spoken of as Professor Senn's. The objections long urged against this procedure are that it leaves a large open wound, which requires weeks to heal, and that the removal of the gauze, after eight, nine, or ten days, can be effected only after the patient has been anesthetized—as to do it without ether is excruciatingly painful. I have modified the operation by sewing together gauze slings with catgut, and placing the suture-line in front of the replaced kidney. I also partly close the wound in the back and put in several additional sutures, which are left untied. When it is desired to remove the slings a nurse stands by the bed, and during the course of half an hour or longer drips salt solution into the wound. When the slings are pulled upon the catgut lets them come apart. One is not obliged to pull any long end around the pole of the kidney, and the gauze is removed quickly and with very little pain. After the removal of the gauze, a small piece is introduced for drainage. The stitches that were inserted at the time of the operation, but left untied, are now fastened; the stitches that were tied at the time of the operation are taken out. As a rule, the wound in such a case should be healed in from eighteen to twenty-one days. I am convinced that if an operation is done at all, some such procedure as this is the proper one. In conclusion, I would say that I operate on fewer cases of movable kidney every year, and treat more of them every year by conservative methods.

The Treatment of Surgical Affections of the Kidney.

By Edward Martin, M.D.,
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The surgical treatment of the various forms of non-infective nephritis, concerning which there has been a recent wave of enthusiasm, can be briefly dismissed by the statement that decapsulation with the idea of establishing a permanent collateral circulation has been shown to be futile, and that simple nephrotomy has so often seemed beneficial in cases of complete suppression of urine incident to sudden congestion or occurring in the terminal stage of acute parenchymatous nephritis that this operation may be regarded as justifiable as a final measure, since it can be rapidly and safely accomplished, often without anesthesia, or, if pain consciousness be preserved, under local infiltration.

To those familiar with the suddenness with which kidney patients apparently well die, and the equal suddenness with which those apparently dying recover, there is

some hesitation in accepting a few brilliant results as proving the efficiency of a given treatment. That nephrectomy has been followed by such results is undoubted. That such results can be reasonably expected remains to be proven by wider clinical experience.

The evidence in favor of nephrotomy for the relief of nephralgia which has reached a crippling degree of intensity is more convincing. Moreover, operation will usually demonstrate the presence of an adequate cause for such nephralgia.

The same statement holds true in regard to renal hemorrhage of such severity or long continuance as to threaten life. This, if not due to traumatism, neoplasm, calculus, tuberculosis, renal displacement, or hemophilia, is usually an expression of nephritis, which, if not cured by the intravenous injection of human blood serum, calls for an exploratory and usually a curative nephrotomy.

The renal incision for the relief of medical conditions should be placed in the avascular line if this can be found, and should be carried to a depth not greater than half an inch, except in the case of profuse and recurrent renal bleeding, when the pelvis should be opened and the kidney thoroughly explored.

The surgical conditions which call for intervention are, in the order of frequency, calculus, tuberculosis, acute and subacute infections of other forms, both renal and perirenal, excessive mobility, new growths, and ureteral obstruction.

The success of surgical intervention is directly proportionate to the timeliness of surgical treatment, and the lessened mortality in renal surgery, noted in clinics the world through, is due far more to early diagnosis and prompt intervention than to improvement in either operative technique or preparatory or after treatment.

The most satisfactory incision is one starting at the angle made by the crossing of the erector spinæ muscle over the last rib, the cut running obliquely downward and forward toward a point half an inch above the anterior superior spine of the ilium; thence, if more room is required for ureteral exploration or intervention, downward parallel to Poupart's ligament. For simple exploration this incision need be very short. For nephrectomy it is sometimes advisable to carry it more directly toward the umbilicus, severing, if necessary, the rectus muscle.

The position which usually makes the kidney most accessible is that suggested by Edebohls—*i.e.*, in ventral decubitus on an air-cushion 10 inches in diameter when inflated, placed beneath the epigastrium. To allow proper chest action the shoulders should be elevated by sand-bags placed on the table beneath them.

For simple nephrotomy designed for the relief of congestion, as in the terminal stages of nephritis, a cocaine anesthesia is satisfactory. Operations implying stripping of the fatty investment and delivery of the kidney through the external muscular wound call for a general anesthetic. Ether is to be preferred, given in minimum doses. This implies a skilled anesthetist, often the preliminary administration of morphine hypodermically, and the beginning of anesthesia by nitrous oxide.

The delivery of the kidney into the external wound may be easy, difficult, or impossible. A failure to realize the last fact has more than once resulted in the sacrifice of an organ which should have been saved, one enthusiastic and experienced operator having given to a reluctant kidney an impulse so forceful that the organ was propelled entirely across the arena.

For the relief of nephrolithiasis the recent habit of surgeons has been to reach the stone by an incision through the kidney cortex placed in its midportion about 30 centimeters nearer its posterior than its anterior surface, and carried thence directly toward the kidney pelvis. This is the usual position of the avascular area, and is sometimes marked by Brödel's white line. This line is often indistinguishable; and the avascular area may be in the middle line or sufficiently displaced to cause the incision to be attended by profuse hemorrhage. It is therefore well before cutting into the kidney to have its pedicle

under control, preferably by the fingers of an assistant. Where the stone is placed in a not markedly dilated renal pelvis, incision through the renal substance seems an unnecessary traumatism which can be avoided by cutting directly through the posterior pelvic wall. In two recent cases thus relieved of renal stone, followed by suture of the pelvic wound, recovery was unusually prompt, nor was there any leakage of urine. Marked dilatation of the pelvis of the kidney always implies ureteral obstruction; if this be due to cicatricial contraction or causes other than the mechanical blocking of the calculus the removal of the latter through the pelvic wall is surely followed by fistula. Such obstruction should, however, be removed. Its detection and treatment are facilitated by a pelvic incision; nor should the operation of nephrolithiasis be considered complete till the patulousness of the ureter is assured by the passage into the bladder of a full-sized catheter (12 F.).

Stones placed in the calyces are best reached through an incision in the parenchyma. Fortunately the *x*-ray enables us to detect both the number, size, and position of the stones. Hence the tedious and often inefficient probing of the various recesses of the kidney is no longer needful.

If it be recognized that in its early stage tuberculous nephritis is often characterized by a dull and intermitting pain referred to the upper lumbar region, by frequent micturition, polyuria, slight excess of leucocytes in the urine, and occasionally microscopic blood, surgical intervention will begin with an effort to determine the tuberculous nature of the lesion and in which kidney it is placed, since it is usually unilateral.

With the diagnostic phase of the question Dr. Noble has dealt exhaustively. When it has been clearly proven that one kidney is tubercular, the other remaining healthy, in the absence of widely disseminated lesions, the indications are for nephrectomy and removal of the corresponding ureter. The operation is difficult and dangerous only when it is complicated by mixed infection, perirenal involvement, and systemic intoxication. These conditions almost inevitably arise in the absence of surgical intervention. The vesical lesions secondary to tuberculous nephritis can usually be cured after removal of the infecting focus.

Acute and subacute infections of the kidney unattended by pelvic retention are best treated by urinary antiseptics. When, however, a pyonephrosis develops this clearly evidences imperfect drainage through the ureter. In cases of prostatics and those suffering from degenerative spinal lesions the obstruction is usually vesical, and betterment follows relief of bladder retention. Renal operation is in such cases rarely indicated. Pyonephrosis of pregnancy usually subsides after childbirth. A pus collection in the kidney not drained by the ureter calls for incision as soon as the assured diagnosis can be made.

Excessive mobility of the kidney, particularly the right, is so common in thin, long-bodied, neurasthenic women that failure to detect it seems almost abnormal. In the great majority of cases it causes no symptoms and does no harm. When, however, it is associated with a constant or intermittently swollen and tender kidney, and particularly when it is associated with recurring attacks of renal colic, the indications for operation are imperative, if a careful trial of abdominal gymnastics, general sanitary measures, and the wearing of a properly fitting straight-front corset are unavailing.

Of the many operations splitting and turning back of the capsule and its suture to the lumbar aponeurosis is a simple procedure which seems to have given as good results as any. Four to six weeks in the dorsal decubitus, with supporting treatment, are curative factors almost as important as the operation itself.

Ureteral obstruction characterized by uro- or pyonephrosis, when due to irregular pelvic implantation or valve formation, may be remedied by plastic operation or by insertion of the cut ureter into the lowest part of the dilated pelvis. When the obstruction is placed near the bladder the ureter may be cut and implanted in this viscus. In the

case of extensive hydronephrosis reaching well down into the pelvis direct anastomosis between the hydronephrotic sac and the bladder has been suggested. An irremediable uronephrosis calls for nephrectomy.

The surgery of new growths, which are nearly always malignant, remains one of almost hopelessness; this because there are no early diagnostic signs. Profuse, causeless, intermittent hemorrhage of renal origin should always suggest neoplasm. The operation consists of complete enucleation through an incision which gives the freest exposure, since these growths are extremely vascular and often adherent.

Uremia.[1]

By James M. Anders, M.D., LL.D.,
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The subject may be considered under three heads: (1) Pathogenesis of uremia; (2) the symptomatology and diagnosis of the condition; and (3) the treatment of uremia.

1. *Etiology and Pathogenesis of Uremia.*—It is generally conceded that the urea excretion is decreased in uremia, although it does not of necessity follow that the condition is dependent upon its retention in the blood and other tissues. Perhaps the opinion that uremia is due to self-poisoning from toxic substances which are normally excreted by the kidneys—*e.g.*, urea, saline constituents, and nitrogenous extractives—was the one most widely accepted in the past. According to most recent writers (Senator, Strauss, Webster, and others) the so-called retention theory, however, is no longer tenable.

Hughes and Carter,[2] from a critical, deliberate study of the subject, reached the conclusion that the condition is caused by an albuminous product unlike anything found in natural urine. Uremia may develop in many cases without an abnormal accumulation of excrementitious materials in the blood. Not only so, but this condition may be encountered, though rarely, in cases in which the nitrogen output is in excess of the nitrogen intake. Achard and Paisseau[1] have shown that the retained urea is most certainly not the cause of uremia, but these investigators contend that the excess of urea in the blood is merely the witness of the retention of other bodies more toxic than the urea itself.

The primary effect of the retained urea is to induce concentration of the blood, followed by a regulating dilution with increased vascular tension. A further change in some cases at least is an escape of the urea into the adjacent tissue; this calls forth a secondary retention of sodium chloride, which tends to counteract, from a physicochemical point of view, the poisonous effect of an excess of urea. While urea is only slightly toxic, and in increased amount in the blood plays no part in the causation of the associated morbid phenomena known as uremia, it is possible that retention of the mass of excrementitious substances due to perverted metabolism or deficient renal function is the principal factor. Pathologists hold that the extractives constitute 57 per cent of the toxicity of the blood in uremia (Fox)[2]. That an abnormal product of a faulty metabolism of the proteids plays an important etiologic part will be more evident hereafter.

Brown-Séquard has advanced the view that the kidneys produce an essential "internal secretion," which function is disturbed in chronic nephritis. The experimental evidence of Bradford goes to show that the kidneys influence markedly tissue metabolism, especially that of the muscles. No pathogenic connection between a diminished "internal secretion" of renal origin and the symptomatic phenomena of uremia can be shown to exist.

Croftan[3] has pointed out that uremia may be due to non-formation of urea. This position is supported by the fact that

[1] Read before the Medical Society of the State of Pennsylvania at Reading, Pa., Sept. 26, 1907.

[1] *Semaine Médicale*, Paris, June, 1904.

[2] *Amer. Jour. Med. Sciences*, August, 1894.

[2] "Thoughts on Uremia," *American Practitioner and News*, vol. 40, 1906, pp. 407-413.

[3] "An Analytic Study of Uremia, with Some General Conclusions in Regard to its Causes and Treatment," *Jour. Amer. Med. Assn.*, Jan. 6, 1906.

"with a decrease of the urea in the urine and blood we very often find an increase of the ammonia." Obviously, then, disturbance of the hepatic function is a causative factor, since the conversion of the circulating ammonia into urea takes place for the most part in the liver. The question, therefore, is pertinent: Does the hepatic derangement play the principal rôle in the causation of uremia?

Unfortunately, our knowledge of perverted functional activity of the liver is inadequate to base thereon definite inferences and conclusions. It is known that the purin bases are normally converted into uric acid in the liver, and the latter substance into urea in the same organ. It is also conceded that circulating toxins of various sorts, which produce the characteristic changes of chronic nephritis, likewise affect the nutrition of the hepatic cells with consequent disturbance of the liver function.

While the hypothesis that uremia is of hepatic rather than renal origin bears the stamp of great plausibility, our knowledge of the physicochemical links in the metabolic chain of events is as yet quite imperfect. Clinical testimony, however, points to hepatic insufficiency as a factor in the pathogenesis of chronic Bright's disease. It is probable that poisons developed in the liver and the gastroenteric tract are thrown into the general circulation, and on reaching the kidneys induce degenerative changes in the renal cells. Certain cases of chronic nephritis may be caused in this manner. It is not, however, until excretion of toxic bodies, as the result of the advancing kidney lesion, is deficient that uremic intoxication ensues.

In general terms, however, it may be assumed that interference with albuminous metabolic processes, followed by disturbed hepatic function and renal function, explains the etiology of uremia most satisfactorily. Strauss found both the ammonia in the blood and the retention nitrogen in the hydremic serum markedly increased in the uremic subject. Other observers have repeatedly shown that the extractives and alloxur bodies are decidedly augmented in this condition. The connection between these metabolic disturbances and uremia is well expressed by Croftan: "An acid intoxication must be incriminated with producing many of the fulminating signs of uremic, as well as of diabetic, coma. The increased excretion of ammonia under these circumstances may be explained on the basis of a chronic acidosis, for we know that ammonia is thrown into the circulation whenever abnormal amounts of acids begin to circulate; this is a protective process, inasmuch as it aids in maintaining the normal alkalinity of the body fluids and the alkali content of the cells."

2. *The Symptoms and Diagnosis of Uremia.*—In acute uremia the severest nervous symptoms develop abruptly and terminate fatally within a comparatively brief time. Convulsions and coma with fever, feeble heart action and pulse, dyspnea and pulmonary edema, enter into the clinical picture, which is commonly unmistakable. But even this fulminant form of uremia may be heralded by prodromata, which are too often unheeded or entirely overlooked. These are malaise, uneasiness or extreme restlessness, slight dyspnea, nausea, mental wanderings, somnolence, and headache, the latter two features sometimes in alternation. This grouping of mild symptoms should be the signal for prompt and efficient efforts at elimination of the poisonous toxins.

Unfortunately, an examination of the urine does not furnish positive proof of an approaching outburst of uremic manifestations. Various tests have been employed, the results of which are intended to foreshadow an attack of uremia, but these are unworthy of professional confidence. Emerson, Croftan, and others have found that the urea determination in the urine has not been of any value in predicting an acute attack. On the other hand, Herter found in chronic nephritis the urea-content in the blood to range from three or four to twenty times the normal. Again, the microscopic findings

in uremia, while corroborative, cannot be relied upon for diagnosis. The amount of renal epithelium found in the uremic urine is generally small, and at the commencement of a seizure tube casts are commonly scarce. Indeed, I have noted the absence of both albumin and tube casts, with a normal specific gravity, in a few cases in which the symptomatic indications of uremic intoxication appeared. In the absence of any crucial test by which to determine the functional capacity of the kidneys, I earnestly desire to emphasize the grouping of clinical features mentioned above, as a means of recognizing the mild forms of uremic intoxication. In severe, acute uremia an assured diagnosis is easily possible and the condition often well characterized and unequivocal.

In chronic uremia the symptoms are complex, extremely varied, and ofttimes indefinite. In general, however, the marked symptoms of acute uremia are conspicuous by their absence, the milder phenomena of the condition alone appearing, forming a late complication of chronic nephritis and lasting over a considerable period of time. Prostration, more or less marked, feeble heart action, irregularly hurried breathing, transient stupor, often alternating with delirium, slight headache, temporary visual disturbance, nausea and anorexia, and muscular twitching make up a grouping of features commonly observed. From time to time the symptoms that characterize acute uremia—the convulsions in particular—may be superadded, and then the immediate outlook at once becomes quite gloomy.

To gain a thorough knowledge of uremia, a divisional study of its symptomatology is demanded, but the scope of my paper will not permit of a detailed discussion of more than a few leading features. An almost constant symptom of uremia is so-called "uremic" or "renal asthma." It often attends early and may be slight and continuous for a long time in chronic uremia. The well-known nocturnal dyspneic attacks are often confused with ordinary spasmodic asthma, their renal origin being overlooked. In uremic dyspnea, however, the respirations are deep and stertorous, or they may be irregular, accelerated, and shallow, sometimes assuming the Cheyne-Stokes type as in acute uremia. The principal cause of the uremic dyspnea is probably the toxemia affecting the respiratory nervous centers, but for therapeutic reasons it is important to recollect that it may result from cardiac weakness, or pulmonary edema.

The nervous phenomena, especially the convulsions and coma, are often conspicuous in the clinical picture, but the fact remains that in most cases certain nervous symptoms, varying with the individual case, are evidenced for a long period without the intervention of the major accidents of this serious condition. I refer to a class of cases in which one finds certain cerebral symptoms, as headache, tremors, alternating exacerbations of delirium, which may be noisy or even almost maniacal (with delusions of persecution at times), in association with the rather slow, hard pulse of the arteriosclerotic type of chronic nephritis. In another group of cases met with, headache and the evidence of acute indigestion, due to overeating, or the partaking of improper foods, with vomiting, foul breath, and sometimes red, swollen lips, gums and tongue, may precede, and recur at irregular intervals, for months or even years, the more serious accidents of the condition.

The *diagnosis* should be arrived at only after a judicious balancing of the data entering into the previous history of the patient, and the closest scrutiny of all the symptoms and the critical and deliberate exclusion of other morbid conditions with which uremia is liable to be confused. Indeed, it may be stated that in the absence of the characteristic urinary findings of chronic nephritis with gross cardiovascular lesions, individual cases may not permit of a clear and accurate discrimination. But even in cases in which the chemist fails to find in the urine the evidences of gross renal lesions, we are sometimes able to render a diagnosis with a

certain degree of probability by a process of exclusion. Indeed, so far as concerns the nervous symptoms of cases of nephritis, it should be an invariable rule, as pointed out by Leube, to ascribe them to uremic intoxication only after all other causes for the same have been excluded. In this same manner we should also endeavor to recognize the pre-uremic state, and mild, evanescent forms of the disorder.

It is undeniably true, however, that the clinician who in the routine examination of his patients ignores the laboratory methods so well known to determine the condition of the kidneys must often fail to interpret the prodromata of uremia, and the same is true of a large proportion of the cases of chronic uremia. It is practically impossible in view of the nature of the symptoms and pathologic processes, without a chemical and microscopic investigation into the condition of the urine, to arrive at a certain diagnosis. On the other hand, negative urinary findings do not justify an exclusion of uremia. Stevens[1] noted an exaggeration of tendon reflexes, especially the knee-jerks and ankle-clonus, and suggests that this may be a symptom of considerable diagnostic import.

There is a group of cerebral affections which are constantly being confused with uremia, and the most important of these is apoplexy. In apoplexy, however, the convulsions occur late, in uremia they as a rule occur early, preceding the coma, which is sudden and deep in the former, while it may be of gradual development in the latter. Complete hemiplegia is known to occur; it may also be a concomitant of uremia, although it is more likely to be transitory in character. Thus in 10 out of 29 cases of uremic aphasia, reported by David Riesman,[2] hemiplegia occurred. Convulsions may precede the right-sided paralysis, but rarely, as in Hopp's case, they follow the development of aphasia. T. H. Weisenburg[3] reports two cases of uremic hemiplegia attended with alteration in the nerve cells of the brain and spinal cord, associated in one with recent primary degeneration of the central motor tract. The uremic nature of these cases was ascertained by necropsy. Obviously, a discrimination is sometimes impossible during the life of the patient.

Finally, the renal origin of hemiplegia may be inferred, if the urinary findings be those belonging to chronic interstitial nephritis (the urine has to be withdrawn), and especially so if the breath emits a urinous odor and if albuminuric retinitis or dropsy be present. Since cardiac hypertrophy and hardened arteries are common to both conditions, it is clear that a concurrence of these serious states may rarely be encountered.

The occurrence of well-developed acute uremia secondary to the acute infections is to be noted. Herter met with some 14 cases of acute lobar pneumonia in which the end presented the symptoms of the acute uremia, and I have recently seen an instance of the sort. A male, aged forty, afflicted with pneumonia, developed decided stupor, the tongue was heavily coated and dry, the pulse rapid and feeble, and there were muscular twitchings. The urinary findings were those of acute parenchymatous nephritis, although the condition of the kidneys prior to the attack of the pneumonia was unknown. After these symptoms had lasted thirty-six hours a general convulsion occurred. The temperature declined by lysis, and before it reached the normal level the patient had had five moderately severe, general convulsive attacks. A slow convalescence followed.

On the other hand, chronic uremia may closely simulate the picture of severe forms of the acute infections, more especially typhoid fever. Without stopping to reoutline the clinical features of uremia in detail, suffice it to direct attention to a few points of similarity, namely, the prolonged torpor or even actual coma; fever and rapid, weak pulse; heavily coated, dry tongue; and the slight muscular twitch-

[1] *British Medical Journal*, Jan. 16, 1904.

[2] "Uremic Aphasia," *Journal of the American Medical Association*, Oct. 11, 1902.

[3] Proceedings of the Pathological Society of Philadelphia, February, 1904, p. 62.

ings. Fortunately, it is possible to distinguish typhoid fever and other acute infections by means of laboratory tests, by the results of a physical examination, and by noting the symptoms and course.

Uremic coma must also be differentiated from opium poisoning and diabetic coma; this can readily be accomplished in most instances by learning the anamnesis, by carrying out a careful urine analysis, and finally, by an ophthalmoscopic examination in suspected cases of uremic intoxication. Osler states that patients in chronic uremia succumb to what he has called terminal infections—acute peritonitis, pericarditis, pleurisy, meningitis, or endocarditis. According to my observation, an acute uremia may also develop in the course of, and secondary to, these terminal conditions when they are grafted upon chronic interstitial nephritis, and cause speedy death.

Treatment.—One of the prime objects of treatment is to meet the dietetic, therapeutic, and hygienic indications presented by the underlying acute or chronic nephritis when evidenced by the characteristic symptoms in the given case.

As elsewhere stated, the diet exerts an important influence upon the course and symptomatology, more especially the nervous phenomena of the disease, thus postponing if not preventing the later development of true contracted kidney. In chronic nephritis, a dietary that will meet the requirements of nutrition, plus the loss of albumen that takes place through the kidneys, ranging from one to three drachms daily, should be adopted, but this limit should not be exceeded. On the other hand, the unfavorable effects of an insufficient diet react prejudicially upon the kidney lesions, hastening, rather than postponing, the final stage with its serious complications, including uremia. As will appear hereafter in uremic, and even preuremic, states a greater restriction of diet is indicated.

Any plan of treatment of uremia that does not include prophylactic measures must be considered incomplete. This dictum also presupposes the recognition by the clinician of the premonitory stage, during which there may be noted a variable number of the following symptoms: uneasiness, mental wandering, delirium, nausea, a dry, heavily coated tongue, foul breath, headache, and a tense pulse. These milder manifestations are a signal for diaphoresis, catharsis, and diuresis of the most prompt and efficient sort.

Additionally, nitroglycerin is to be employed freely; it usually brings about a reduction of the vascular tension and renders the second aortic sound less accentuated. In cases in which nitroglycerin fails to decrease satisfactorily the arterial tension, I have found it highly advantageous to combine aconite, which lowers blood-pressure in acute uremia, while at the same time exercising a diuretic and diaphoretic influence. The dosage must be proportioned to the degree of abnormal pressure, but even in cases in which the vascular tension was high, one-minim doses of nitroglycerin combined with two-minim doses of tincture of aconiti radicis, administered every third hour, proved signally successful. Unfortunately, it is, as a rule, impossible to effect elimination through the kidneys owing to the presence of advanced structural changes.

Robinson[1] is inclined to order repeated doses of sweet spirit of nitre, or to add a teaspoonful to a tumbler of water and request the patient to drink a mouthful of it every hour or two to excite diaphoresis. He continues: "The nitre relaxes the arteries (as nitroglycerin and the nitrites), acts as a diaphoretic, and is effective and innocent." I can confirm this statement from my own recent experience. Hot wet packs, or the dry hot-air bath, usually induce copious sweating and prompt elimination of the unknown poisons through the skin. It has been claimed that the electric light bath possesses a decided advantage over the hot packs and dry hot-air baths, in that it promptly induces dia-

[1] "The General Management and Therapeutics of Nephritis," *American Journal of the Medical Sciences*, July, 1904.

phoresis without elevating the temperature of the surrounding air; also that there is greater elimination through the skin and no prostration, so that it may be continued for a long time. Saline laxatives form the best means of accomplishing catharsis: they are to be given in concentrated solution when the stomach is fasting.

Elaterium, or preferably its neutral principle, elaterin, in the form of trituratio elaterini (dose ¼ to 1 grain), may also be used; it exerts prompt action and produces large, watery stools. When the patient is comatose and cannot swallow, intestinal elimination is accomplished with great difficulty.

Pilocarpine has many warm advocates, and its efficacy as an eliminant is undoubted since it acts upon the entire human secretory apparatus. The most eligible preparation is pilocarpine hydrochlorate, which may be administered hypodermically, the dose ranging from ⅛ to ¼ grain. My experience of the use of this remedy is quite limited, and I find myself in accord with the views so well expressed by Forchheimer regarding its employment: "The danger in its use, especially in children, has prevented me from administering this drug in a routine way, and I reserve it for those conditions in which the other diaphoretics and the cathartics do not produce the required effects. I should certainly hesitate to give pilocarpine when there is any danger of development of pulmonary edema, nor should I be consoled if such edema did occur, even though I had added atropine to it."

The diet is to be restricted to articles that give rise to the smallest accumulation in the blood of residual nitrogenous bodies. On this ground I hold it to be all-important to allow only the blandest fluid foods, as skimmed milk, or buttermilk, or even whole milk, preferably hot. In chronic uremia the dieting does not need to be so severely rigorous as in the acute form, and must be determined for the individual case.

It is true, as has been claimed, that there is no precise evidence to show that the sweat or the urine in uremia, after the use of diaphoretics and diuretics, contain any of the albuminoid or alkaloid bodies that must be accused of causing the distressing symptoms of the condition, but I feel strongly that serious and even dangerous uremic manifestations can be averted by the general plan of management recommended above.

When the milder manifestations are not thus controlled, and in all cases of either uremic convulsions or actual coma, heroic measures must, in addition, be brought promptly into requisition. Uremic coma calls loudly for active catharsis. In cases in which high tension is associated, it must be quickly reduced. If the patient in convulsions be robust and plethoric, venesection, withdrawing from 16 to 30 ounces of blood, should be practiced. This measure diminishes the toxicity of the blood, and its efficacy is greatly enhanced by an intravenous counter-injection of large quantities of normal salt solution, which further reduces the toxicity of the blood by diluting it. As a result of these combined measures—venesection and injection of saline solution—cures have been reported, and I have observed one case apparently moribund in which recovery ensued. The operation of bloodletting may be repeated at the end of twenty-four hours if deemed necessary.

The question whether morphine should be prescribed in cases of uremic convulsions is still subject to discussion, although the balance of authority is in favor of its employment. It is generally held to be effective in lessening the number and severity of convulsive seizures. MacKenzie, to whom we owe its introduction as a remedy in uremic convulsions, the late A. L. Loomis, Osler, Delafield, and others, have all warmly advocated its use. Tyson holds that it produces harmful effects in the convulsions of chronic interstitial nephritis, and with this view I am inclined to agree. On the other hand, in acute nephritis and the chronic parenchymatous variety I have witnessed excellent results from its use, hypodermically.

Chloroform and ether inhalations are worthy of a thorough trial in extreme cases, but are less successful than morphine appropriately exhibited. The one leading objection to chloroform and ether is that their effects are too evanescent. Neither do they meet the important indications arising from the etiologic conditions. The convulsions can also be controlled by chloral given by the mouth or by the rectum, in dosage of from 10 to 20 grains if administered by the mouth, or 30 to 40 grains per rectum. This dosage must be repeated, first at half-hour intervals, afterward at one- to two-hour intervals, promptly withdrawing the remedy, however, as soon as the spasms are controlled.

Le Fevre[1] has pointed out that the advantage of chloral over chloroform is that it has a more lasting action, although it merely antagonizes the effects of the causative toxins upon the nervous system.

For the dyspnea which attends and is spasmodic in character and origin, I would advise antispasmodics and nervous sedatives. Respiratory stimulants, as strychnine, atropine, and the like, have failed in my experience to meet this symptomatic indication.

The gastrointestinal group of symptoms may demand the physician's attention. It is to be recollected that they merely indicate an effort on the part of nature to rid the system of toxins. This tendency to self-elimination should be encouraged by lavage, both gastric and intestinal. Astringents are not permissible, but purgatives may be employed, for the double purpose of removing the toxins formed in the intestines and averting their absorption into the general circulation. Obviously, no attempt should be made to arrest a uremic diarrhea.

The treatment as above described is intended to serve as a general guide; it will not be found to be universally applicable, since the symptomatic indications manifest wide differences in different cases. With all our vast hygienic and therapeutic resources, there should be scope for real achievement in the treatment of uremia, but seriously considered the triumphs of our profession in the past and present in this direction are not desirable auguries for the future.

[1]"The Treatment of Uremia," *British Medical Journal*, Nov. 24, 1906.

The Eye and the Kidney.[1]

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The relation of diseases of the kidneys to certain lesions in the fundus of the eye is well recognized; generally these lesions are somewhat loosely grouped under the term "albuminuric retinitis." The object of the present communication is to endeavor to show that this relationship from the diagnostic and prognostic standpoint is a more important one than is usually recognized by many clinicians, that the lesions in the fundus oculi coincident with various types of renal disease do not manifest themselves only in the form of "albuminuric retinitis and its varieties," and to present briefly an account of those ocular manifestations which may be associated with or caused by various forms of Bright's disease, dwelling, however, particularly on those which are of interest to the general clinician. I proceed at once to recount the ocular lesions just referred to; they are:

1. Complete blindness without ophthalmoscopic lesions, or at least without the presence of lesions more or less suggestive of disease of the kidneys, generally called uremic amaurosis, and most often seen in acute nephritis, but also in acute exacerbations of chronic renal disease.

2. Various types of retinitis and neuroretinitis, to which the descriptive term

[1]Read at the fifty-seventh annual session of the Medical Society of the State of Pennsylvania.

Certain other publications by the author on this subject have been freely utilized in the preparation of this paper, notably "Certain Changes in the Vessels and Vascular Coats of the Eye," *Maryland Medical Journal*, June, 1900; "The Ocular Manifestations of Chronic Bright's Disease," *Medicine*, December, 1902; "Concerning the Signs in the Retina of Persistent High Arterial Tension and Their Diagnostic and Prognostic Import," *Ophthalmic Record*, August, 1906; "Intraocular Angiosclerosis and its Prognostic and Diagnostic Significance," International Clinics, vol. i, 17th series, 1907.

"albuminuric" is commonly applied, and which are most often seen in association with chronic forms of kidney disease.

3. Alterations in the caliber and relation of the retinal vessels owing to sclerotic changes in their walls, with or without hemorrhages and exudates in the retina, seen in association with those forms of renal disease in which vascular changes are evident elsewhere in the body; also isolated hemorrhages and exudates, without marked vessel-wall changes.

4. Alterations in the uveal tract, particularly in the choroid and iris.

5. Some varieties of cataract.

6. Paresis and paralysis of the ocular muscles, particularly the superior oblique and the external rectus.

7. Recurring subconjunctival hemorrhages.

Uremic amaurosis, common in acute nephritis, particularly in the nephritis associated with scarlet fever and pregnancy, may also occur in chronic nephritis, which has already produced organic changes in the retina. Indeed, it may occur, as Lawford has well said, in any of the various forms of renal disease in which there is sufficient interference with excretion by the kidneys to load the blood with those poisonous materials, whatever their exact nature may be, which induce the uremic condition. What particularly represents the constitution of these poisonous materials, and what the relation of interference with albuminous metabolic processes and disturbed hepatic and renal functions bears to the etiology of uremia, has already been described in a most admirable manner by my distinguished colleague, Dr. Anders.

In uremic amaurosis the loss of sight usually is sudden, bilateral, and sometimes complete; more rarely the visual disturbance comes on gradually. The pupils are irresponsive to light stimulus in some patients; in others they react normally. With the ophthalmoscope no distinct lesions may be discoverable, or only a slight edema of the retina, except in those cases in which a coexisting chronic nephritis has already produced organic changes in the retina. Whether the blindness is due to a toxin acting on the cortical centers, or to intoxication of the retinal elements, is in dispute. Many clinicians, as well as ophthalmologists, believe that the failure in vision depends upon the action of a toxin directly upon the ganglion cells of the retina. More recently, however, investigations seem to show that it is really the cortical centers upon which the poisonous action falls, proved by the fact that in some of these cases, when sufficient vision has been recovered to make the test, a typical homonymous hemianopsia is present, which could not be explained by the presence of a retinal lesion.[1] So also retention of the pupilary reflex with absolute blindness indicates that the lesion in this condition resides in some portion of the visual pathway beyond the primary optic centers.

The blindness may last from a few hours to a day or more, and the return of sight is usually comparatively sudden, and the condition, except in rare instances, does not lead to permanent blindness, although, as Leber has shown, very occasionally it seems to be the cause of an optic nerve atrophy which develops some time after its subsidence. This, it would seem, indicates that some of the cases are not cortical in origin, but depend upon a retinal ganglion cell intoxication which has lasted sufficiently long to produce a permanent change in the optic nerve elements, precisely as it does after certain other poisons, notably the toxin of diabetes, methyl-alcohol, large doses of quinine, etc.

Retinitis.—That some disturbance of sight may occur in the course of the various types of Bright's disease was known to Bright himself, and was studied pathologically in preophthalmoscopic days. Five years after the discovery of the ophthalmoscope the lesions visible with this in-

[1]Personal communication from Professor Pick, of Prague.

strument were described by Heymann and later figured by Liebreich in his well known Atlas, in the form of a water-color which has since found its way in reproductions in almost all publications on this subject.

The so-called neuroretinitis of Bright's disease may appear as a typical albuminuric retinitis, a degenerative albuminuric retinitis, a hemorrhagic albuminuric retinitis, an albuminuric neuroretinitis, and an albuminuric papillitis.

The typical albuminuric retinitis manifests itself in various shaped and placed white dots in the macula and its immediate neighborhood, sometimes so arranged that a star-shaped figure, or macular figure, is produced, while around the papilla are large, yellowish-white areas forming a striking white region, to which the name "snow bank of the retina" has often been applied. Linear and flame-shaped hemorrhages are scattered over the eye-ground, and particularly noteworthy in the fiber layer of the retina and along the blood-vessels, which in their turn may be buried in the white exudate or pass over it. Three stages are usually present, indicated by the names hyperemic, degenerative, and atrophic.

In the degenerative albuminuric retinitis the white spots are separated by normal areas, the hemorrhages are less conspicuous, and the arteries show patches of sclerosis in their walls. The disk is unduly red, and the star-shaped figure is either wanting or is replaced by white spots dotted irregularly over the macular area. As time progresses the disk assumes a dirty-white, atrophic hue, the vessels grow narrower, and secondary pigment areas appear in the retina. In the hemorrhagic albuminuric retinitis and albuminuric neuroretinitis and papillitis the conspicuous features are in the one hemorrhages, particularly around the papilla and in the nerve fiber layer, and in the other marked swelling with edema and inflammatory signs which swell forward the nerve-head until it assumes the appearance typical of papillitis or choked disk. While systematic writers have recognized these types, they often shade one into the other, and for practical purposes it is sufficient to recognize two great varieties, usually termed inflammatory or exudative, and degenerative.

It is a matter of great importance to determine the earliest signs of renal retinitis, and these may consist in small, insignificant white dots or fluffy exudates, a curious brick-red discoloration of the optic papilla, and alterations in the caliber and walls of the retinal vessels. If the macula is early and much involved, there is also early depreciation of vision, but it must be remembered that disturbance of sight, especially in those cases in which the periphery of the retina is first attacked, is not a conspicuous sign, and it is quite possible to have for long periods of time good visual acuity in so far as test-letters are concerned, even when there is an extensive renal retinitis, and therefore a mere test of vision during the course of Bright's disease is no indication of what the actual lesions may be unless elaborate and careful ophthalmoscopic examination is made. Scotomas, blue-blind areas, alterations in the light-sense, blue-yellow blindness, and, as has been pointed out by Simon, violet-blindness, are not uncommon symptoms in albuminuric retinitis, and the last named disturbance of color-sense, as well as blue-blindness, first described by Gerhardt, have been thought to be more or less characteristic of the disease. As serious late complications, detachment of the retina, hemorrhagic glaucoma, and elaboration of retinal hemorrhages may occur, as well as occasional stoppage of the central artery of the retina, retrobulbar neuritis, and hemorrhages in the sheath of the optic nerve.

Varieties of Renal Disease which Produce Retinal Lesions.—In general terms it may be said that ocular lesions may occur with any form of nephritis, but there is no question that the various types of retinitis

are most frequently associated with chronic interstitial nephritis, that is with renal sclerosis, whether this is an independent primary affection or whether it is a sequel of arteriosclerosis. Various types of retinitis and retinal lesions may be symptoms of chronic parenchymatous nephritis. Some observers, for example Samuel West, make a sharp distinction between the degenerative and exudative forms of albuminuric retinitis, believing that the former is indicative of chronic granular nephritis, and that the degenerative changes depend upon vascular alterations, while the latter indicates a chronic parenchymatous nephritis and depends upon an inflammatory or toxic influence.

Naturally, the so-called secondary contracted kidney, which is a sequence of the large white kidney, may be associated with retinitis. The statement is frequently made that amyloid disease of the kidney produces albuminuric retinitis, and there is no reason why it should not, because lardaceous degeneration of the kidneys is not a separate disease, but purely an event in the course of Bright's disease, most frequently, if I mistake not, occurring in parenchymatous nephritis of cachectic origin. Although uremic amaurosis, to which reference has already been made, is generally seen with acute forms of Bright's disease, there is no reason why an amaurosis of this character should not take place, as, indeed, it not infrequently does take place, during the course of a retinitis caused by chronic kidney disease, as the result, for example, of an exacerbation of the renal lesions.

Date of Occurrence of the Retinal Lesions.—At one time it was supposed that the retinal lesions of renal disease began at the same period that cardiac hypertrophy was manifest, but this certainly is not a constant association. In general terms it is probable that the renal disease must be present for some months before the retinal lesions appear, but the very fact that retinitis may be the first sign of kidney disease, indeed, as already noted, the means by which it is discovered, renders an authoritative statement on this subject all the more difficult. There has been a good deal of discussion as to the existence of the so-called prealbuminuric retinitis, and a number of cases have been recorded—that is, the retinitis, retinal hemorrhages, exudates, etc., have been found to precede the albuminuria by a considerable length of time. Dr. Sutton suggests that these are examples of granular kidney in which albumin is absent, or only present to a very small amount until a later stage of the kidney affection. In other words, it is conceivable that the patient with retinal lesions might consult the physician, as Groenouw suggests, during a period when albumin was lacking, and that therefore these lesions might be ascribed to a prealbuminuric period, although really they appeared during a period when albumin is temporarily absent. On the other hand, lesions especially located in the blood-vessels, particularly various types of perivasculitis and of "silver-wire arteries," producing compression upon under- and overlying veins, are often present for long periods before nephritis is manifest, or at least before other signs of renal disease are fully established—that is, albumin and casts.

Age at which Retinal Lesions Occur.—Inasmuch as chronic interstitial nephritis is a rare disease before the twenty-fifth year, its percentage rising between that age and forty and growing still greater between forty and sixty, the retinal lesions are likely to appear at the same periods. According to Nettleship, the age varies from thirty to sixty, the most prolific single decade being from fifty to sixty. Children, however, are not exempt. In other words, the disease may occur at any age. Reference is not now made to uremic amaurosis—for example, in scarlatinal nephritis—but to so-called typical albuminuric retinitis. The youngest case recorded by Bull in his analysis of 103 cases of exudative neuro-

retinitis associated with chronic Bright's disease was five years of age. Arnold Lawson and G. A. Sutherland have observed this affection in a patient twelve years old, and I have seen one case in a child of nine.

About twice as many cases of renal retinitis occur in men as in women, and it is stated that when decided hyalin thickening of the retinal arteries is evident to the ophthalmoscope, an early stage of granular kidney may be suspected, especially if the patient is comparatively young (Nettleship).

Frequency of Retinal Lesions in Chronic Bright's Disease.—To ascertain accurately the exact proportion of renal disease in which retinitis occurs is difficult, and the recorded percentage has varied from nine to thirty-three. Groenouw has collected from the literature 935 cases of kidney disease, among which there were 209 examples of retinal lesions, namely, 22.4 per cent. According to William F. Norris, fully 25 per cent of the cases of chronic Bright's disease as they occurred in general hospitals are affected by various forms of retinitis. My own experience yields an equally great if not greater percentage, and if one includes not alone the cases in which there are more or less typical forms of retinitis and retinal lesions, but also those in which the ophthalmoscopic signs are comparatively insignificant, consisting chiefly of alterations in the walls of the retinal vessels, this percentage will rise much higher. It is interesting and important that Litten among 33 cases of chronic parenchymatous nephritis six times noted albuminuric retinitis.

Course and Prognosis.—In general terms the appearances of the eye-ground in the various types of renal retinitis have been described, and under treatment there may be subsidence of the neuritis, disappearance of the opacities in the retina, and absorption of the hemorrhages, although I am inclined to think that all of the white spots never subside, and that those in the macular region are the most permanent. The serious complications of secondary glaucoma, thrombosis of the vessels, etc., have been referred to. Therefore the prognosis, in so far as sight is concerned, depends largely upon the character and situation of the exudates and hemorrhages. In some of the exudative varieties, with great involvement of the macula, vision is markedly reduced, although at times florid types under proper treatment clear up very rapidly. An absolute disappearance of the lesions is uncommon and perhaps never entirely occurs, and the physician must be constantly on the lookout for fresh exudates and hemorrhages, which of themselves furnish prognostic indications of the course of the disease. The prognosis *quoad vitam*, if the large statistical material now available is studied, is most unfavorable, and the statement that patients with albuminuric retinitis rarely live longer than two years after its detection, and a very considerable percentage of them die within the first year of its development, is much quoted, but there are many exceptions to this rule, and careful records will show that patients have lived five, seven, and even a greater number of years after the retinal lesions have appeared. In this respect there is a marked difference between hospital and private patients, or between those who can afford the best treatment and those who have to take their chances with less careful therapeutic and dietetic surroundings. The late Dr. Belt collected 155 cases from private practice, of which 62 per cent died within one year, 85 per cent of the remainder in two years, and 14 per cent of the remainder lived more than two years. Of the 75 cases collected from hospital practice, 85 per cent died within one year, 93 per cent of the remainder within two years, and 6 per cent of the remainder lived more than two years. Miley's statistics of 164 patients with acute and chronic renal affection showed 105 with unaffected eyes and 51 with retinitis albuminurica, and that the death-rate of patients with the retinitis

was double that of those who were free from this complication. Now, while the prognosis of albuminuric retinitis will always remain grave, I am satisfied that statistics gathered from now on will show an improvement so far as the duration of life is concerned, as compared with those which are already upon record. This depends upon the improvement in our therapeutic methods. It must be remembered that ophthalmologists often see the lesions of renal retinitis late—that is, they probably have existed some time before their detection—and therefore it is not correct to say that the majority of patients die within two years after the appearance of retinal changes in Bright's disease, but only that they are apt to die within two years after these changes have been discovered.

The *pathological anatomy and pathology* of the conditions just described do not, it seems to me, belong with propriety to the present paper, and they therefore may be dismissed by saying that the so-called renal retinitis depends upon an altered condition of the blood, degenerative changes in the blood-vessel walls, and excessive pressure within these vessels. The retinas themselves are thickened owing to the presence of inflammatory edema, and fatty degeneration of exudates and of the retinal elements is responsible for the alterations in the macula. Perhaps the most important alterations from the pathological standpoint are those which occur in the vessels—that is to say, the same causes which originate disease of the blood-vessels of the kidneys are capable of making similar alterations in the vessels of the retina, and to these alterations the chief rôle must be ascribed in causing the various types of retinal lesions. Indeed, no less distinguished an author than von Michel believes that so-called retinitis albuminurica is entirely the result of a disturbance in the circulation and of lesions in the retina originated by primary disease of the vessel system.

Diagonsis.—Although the word "typical" has frequently been used in the description of albuminuric retinitis, it must not be taken in too literal a sense. In other words, appearances exactly simulating retinitis albuminurica may occasionally be found in syphilitic retinitis, in the retinal hemorrhages of young persons, and in some types of anemia. A very interesting and important fact, pointed out many years ago by von Graefe and other observers, is that appearances identical with those of albuminuric retinitis, even to the formation of the macular figure, may be seen in brain tumor. This observation I have confirmed many times in my own experience, and have quite recently recorded some cases of this character. Rarely albuminuric retinitis is unilateral, at least temporarily unilateral, although usually bilateral before the termination of the patient's life. When the appearances described as typical are present and the exceptions already noted are eliminated, the presence of chronic renal disease is strongly suggested. What I wish, however, to particularly point out is that it is a mistake to suppose that eye-ground examination is negative if those lesions which we have already described as typical are absent. In point of fact, the so-called typical cases are the rare ones, or comparatively rare ones, while the types with scattered hemorrhages, irregularly placed exudates, forms of perivasculitis and early changes in the nerve-head and retinal vessel distribution are the ones that are most startling and most important in their significance, and it is to these that I wish now to call particular attention, under the title of

Alterations in the Caliber and Relation of the Retinal Blood-vessels as a Sign of Nephritis.—For their description I take the liberty of quoting from one of my papers:

The suggestive signs include uneven caliber and undue tortuosity of the retinal arteries, increased distinctness of the central light streak, an unsually light color of the breadth of the artery, and alterations in the course and caliber of the veins.

The pathognomonic signs include changes in the size and breadth of the retinal arteries of such character that a beaded appearance is produced; distinct loss of translucency; decided lesions in the arterial walls, consisting of white stripes in the form of perivasculitis; alternate contractions and dilatations of the veins, and particularly—and this is the most important of the signs—indentation of the veins by the stiffened arteries in the same manner as a solid rod would indent a rubber tube where lying across it. Sometimes the vein is simply flattened slightly at the point of crossing, or merely pushed aside, or its caliber is contracted so that beyond the point of crossing there is an ampulliform dilatation. In addition to these well-known signs, there may be changes in the venous walls, so that they are bordered with white stripes, and the veins may be exceedingly tortuous and contain varicosities. Finally, there are edema of the retina in the form of gray opacity around the disk or following the course of the vessels, hemorrhages manifesting themselves as linear extravasations, or roundish infiltrations, or sometimes assuming a drop-like form.

1. *The Age at which These Signs May Occur.*—As Marcus Gunn has said in his well-known papers on this subject, old age alone does not produce these changes, or does not necessarily produce them in their most elaborate form. Perfectly healthy retinal vessels are often visible in persons who have reached old age, even seventy or eighty years of life. Doubtless the visible changes, again to quote Mr. Gunn, usually begin about forty and fifty. I have seen them, however, at an earlier age, one of the most pronounced examples being a man aged thirty-seven years. Referring to intraocular vascular disease and the nature of the pathological processes at work, George Coats remarks that if all changes which involve thickening of the wall and increased rigidity are included in the term angiosclerosis, it is not entirely a pathological process, but in part a senile change which is naturally present in the vessels of old persons, and in part the result of a pathological stimulus. It is difficult always to separate these two components. Given the stimulus, however, he further remarks, the changes may appear far in advance of the usual age, as, for example, in juvenile nephritis. In other words, as I have elsewhere said, if the cause of persistent high arterial tension is sufficiently prolonged, these alterations may occur at any age at which the pathological condition to excite them arises.

2. *The Earliest Indications.*—The suggestive signs have already been enumerated and must not be disregarded if they are associated with other symptoms of arterial disease. Three signs may be seen early: (*a*) A markedly corkscrew appearance of certain arterial twigs, either of those which skirt the macula, or, more significantly, of one or more small branches which arise from the larger vessels of the main distribution which themselves are apparently normal. A particularly striking manifestation is one not infrequently seen of a single twig which descends vertically from a transverse branch and assumes this corkscrew appearance. This manifestation is the one to which Alleman has given the name "crinkled retinal vessels," and which he figures in somewhat exaggerated degree in one of his diagrams. Such phenomena indicate that the whole artery is not affected, made evident, as has been often noted before, especially by Mr. Gunn, not only with the ophthalmoscope, but, where opportunity afforded, by means of the microscope. Thus, Coats points out that serial sections from the small vessels will frequently demonstrate notable differences within a very short space. For example, in the central artery of the retina the region of the lamina is often selected for the endarteritic process, where the vessel may be almost obliterated, although a few millimeters from this position it is practically normal. What has been termed the irregularity of the distribution of vascular disease in the retinal vessels is, therefore,

well known, both from the ophthalmoscopic and the microscopic examinations.

(*b*) A flattening of a vein where it is in contact with an artery, and at this spot it has somewhat the appearance that a strap would have if laid across a solid tube, provided the vein passes over the artery. If the artery passes over the vein, the appearance is analogous to that which would be produced if the strap were placed across the lower surface of the tube. The vein is only slightly compressed at this stage, but is not yet indented sufficiently to produce an ampulliform dilatation of the vein beyond the point of crossing. It has seemed to me that this appearance is a little more frequent in the inferior temporal retinal vessel distribution than elsewhere, or, perhaps, to speak more accurately, appears there first.

(*c*) The nerve-head has an appearance often loosely described as "congested." But this appearance is different from that presented by the so-called streaked hyperopic disk; it is unlike the flannel-red surface of the papilla which merges into an equally flannel-red eye-ground, so commonly the result of prolonged eye-strain, exposure to bright light and intense heat, or seen in certain constitutional conditions; and, finally, may be distinguished from the early stage of neuritis, with the somewhat juicy-red aspect of the disk. It is a dull-red appearance which is presented, and differs from the franker congestions as the unhealthy flush of a cheek differs from the brighter color of a normal blush. Subsequently the later signs develop—marked indentation of veins, with ampulliform dilatation beyond the point of pressure, varicosities, "silver wire" arteries and perivasculitis, hemorrhages, etc.

Naturally, the significance of the signs just recited pertains especially to arteriosclerosis, and, as I have pointed out in other communications, they represent an importance from the diagnostic standpoint quite equal to, if not greater than, that furnished by a study of the pulse, the character of the first sound of the apex and the second sound of the aortic area, the sphygmogram, and the results obtained by instrumental measurement of arterial tension. But I am not using them along these lines at the present moment. I wish to emphasize that exactly similar appearances are of the greatest importance in the investigation of renal conditions and in summing up our general prognosis. Not only should our thoughts be turned to the prognosis with regard to sight, but how far such a disease may be regarded as an indication of the condition of other organs of the body, and not only of the intimate connection between these ophthalmoscopic changes and cerebral vascular disease, but because they have an important relationship to the study of chronic nephritis, and because they are often seen in many cases in which the usual retinitis has not yet developed. As I have elsewhere said, they are but part of the symptomatology of arteriosclerosis which may be associated with or eventuate in renal sclerosis, and as we know that a large percentage of the cases of chronic interstitial nephritis are primarily arterial in causation, this relationship increases in importance. These retinal vessel changes may be discovered where the usual retinitis may not supervene, because it is possible for them to be significant of a process which may terminate fatally before the development of fully established nephritis.

The other lesions which are found in connection with diseases of the kidney, in so far as the eye is concerned, are largely of interest to ophthalmologists, and I will content myself with reciting a few of them, namely, diseases of the uveal tract, particularly in the form of curious types of disseminated choroiditis and choroidal hemorrhage; certain persisting types of iritis, and if iritis is stubborn and unexplained by other pathological factors, thorough urine examination is necessary; some varieties of cataract, the so-called nephritic cataract, and in the presence of cataract urine examination is essential; and, finally, rare types of ocular muscle palsy, notably paralysis of the superior oblique, external rectus, and other orbital muscle palsies. Paresis of the cil-

iary muscle has frequently been observed. Last but not least in significance are recurring subconjunctival hemorrhages and subcutaneous palpebral hemorrhages. Textbooks are fond of referring to the hemorrhagic phenomena of nephritis, epistaxis, purpura, retinal extravasation, etc., but usually forget these slight subconjunctival and subcutaneous palpebral hemorrhages, often occurring in persons past forty, usually during sleep, and more frequently, I think, in the left eye. They come and go, recur at comparatively short intervals, seem to be very insignificant at the time, and yet are often as significant as the deeper retinal lesions of serious arterial disease in association with nephritic changes.

Treatment.—Naturally, the treatment of the conditions which I have described is concerned chiefly with that which is suited to the disease which has caused them. Speaking particularly from the eye standpoint, I would say that the best results in exudative albuminuric retinitis are reached by the administration of repeated doses of alteratives, the various iodides, biniodide and bichloride of mercury, the suitable administration of vapor baths, and if there is any persistent high arterial tension, the exhibition of repeated small doses of nitroglycerin. In a few cases I have thought that very large doses of the various preparations of iron were efficient, especially iron exhibited in connection with the bichloride of mercury. Local treatment is of no importance, and I have failed to gather from a study of the literature, particularly as it has been gathered by Dr. Suker, that decapsulation of the kidney has any favorable influence on the subsidence of the retinal lesions.

Before dismissing this subject I ask your permission to emphasize certain points.

1. While there is a so-called typical albuminuric retinitis, with star-shaped figure in the macula, hemorrhages and exudates scattered throughout the eye-ground, and the so-called snow-bank appearance around the disk, this is by no means the most frequent manifestation of renal retinitis, which is more often exhibited in the form of congestion around the optic nerve, small, scattered hemorrhages and dots in the macula, and fluffy exudates in the periphery of the eye-ground, which might readily escape detection unless the most searching ophthalmoscopic examination is made.

2. That in a certain percentage of cases, in association with other indications of arteriosclerosis, intraocular angiosclerosis, with alteration in the caliber of the retinal vessels, indentation of underlying veins, slight perivascular changes in the form of the so-called "silver-wire" arteries, is deeply significant of a process which may eventuate in a chronic interstitial nephritis, which has not produced the ordinary so-called renal retinitis.

3. The presence of normal vision does not in any sense exclude the existence of the retinal lesions of chronic Bright's disease, and no patient is properly examined until the ophthalmoscope has been used in expert hands, preferably after dilatation of the pupil with a suitable mydriatic. The ophthalmoscopic revelations, already now many times recited, are so important in the study of every case of nephritis, no matter what its type may be, that a systematic and routine and frequently repeated ophthalmoscopic examination of all patients with Bright's disease at suitable intervals should never be neglected, any more than a similar frequently repeated examination of their urine is neglected, not only because a renal retinitis may appear and develop most insidiously, but because by reason of such development and in the course of such examination the prognosis of the case is suitably checked. Naturally, ophthalmoscopic examination appeals to me, not only in this disease but in all other diseases, but I maintain that for the reasons already recited these repeated ophthalmoscopic examinations are just as important as examinations of the blood-pressure, of the urine, and in general terms of the cardiovascular system in various types of chronic interstitial and parenchymatous nephritis.

EDITORIAL.

THE SYMPOSIUM ON NEPHRITIS.

Without doubt one of the most important conditions which is presented, for the consideration of the general practitioner as well as the specialist, is the pathological changes which arise in the kidneys. The busy practitioner not infrequently finds that efforts to procure relief from certain symptoms are nullified by his neglect to pay a sufficient amount of attention to these important eliminating organs. The symposium upon nephritis which we publish in this issue of the THERAPEUTIC GAZETTE will undoubtedly prove of very great value to our readers, as presenting a complete summary of modern views in regard to renal disease, both from the standpoint of the pathologist, the medical clinician, the surgeon, and the ophthalmologist. Each of the contributors is well known in his particular field and speaks with the authority of experience.

NEW METHODS OF INVESTIGATION IN REGARD TO THE EFFECTS OF DRUGS, WITH ESPECIAL REFERENCE TO THE INFLUENCE OF QUININE.

Although it cannot be denied that a very large part of our employment of drugs in the treatment of disease is still based upon empiricism, it is nevertheless a fact that during the last thirty years extraordinary advances have been made in our conception of the manner in which drugs produce many of their therapeutic effects. By experiments carried out with various forms of blood-pressure apparatus we have gained fairly accurate information in regard to the physiological action of drugs which are known to possess a considerable influence upon the circulatory system, and much work has been done in regard to the effects of anesthetics, and the influence of various drugs upon the secretion of bile and the action of the kidneys.

With the advances which have been made in pathological and physiological research, particularly in connection with the conditions of the blood, new fields of therapeutic investigation have been opened, and these are now being tilled in such a way as to produce crops the value of which may well be said to be inestimable, although as yet the growth is so immature that the full value cannot be calculated.

Of all the investigations which have done most toward clearing up our knowledge of infectious diseases and the means by which the body antagonizes such attacks, the work which has been done by Ehrlich and many other investigators upon the condition of the blood is without doubt the most valuable, and these researches in turn have been advanced by the studies of Wright and his associates in regard to the influence of opsonins, so that at the present time we have fairly clear conceptions of the several methods by which the organism protects itself against invading microörganisms.

It having been found that it was possible to increase the ability of the body to protect itself from invasion by infectious germs through the stimulation of the development of antibodies and opsonins and the activity of phagocytes, it at once became evident that a wide field of investigation existed in regard to the effects which drugs might produce upon these protective processes of the body, and it became clear that many of our therapeutic procedures which have heretofore been regarded as of great value, although purely empirical, might rest upon a sound scientific basis.

The readers of the GAZETTE will remember that we have pointed out in its original and editorial columns at various times that many drugs which heretofore have been supposed to do good by acting as stimulants, in the sense of circulatory and respiratory stimulants, might prove to be not stimulants in this sense at all, but rather in the sense that they in-

crease the ability of the phagocytes to devour microörganisms, or the ability of antibodies to combine with the toxins produced by microörganisms, or the ability of the opsonins to aid the corpuscles in destroying invaders. It was on this basis that a research was published by the writer of this editorial four years ago which seemed to show pretty positively that the administration of alcohol in moderate doses during the course of acute infectious diseases distinctly increased the bacteriolytic power of the serum.

Our attention has been called at this time to two very interesting and valuable contributions upon the action of quinine sulphate in the human blood, one of which is from the Hull Physiological Laboratory of the University of Chicago, by Thomas M. Wilson, published in the *Journal of Physiology* of September 2, 1907, and the other a research by Manwaring and Ruh from the Pathological Laboratory of Indiana University, printed in the *Journal of Experimental Medicine* of September 21, 1907. In the first research, that by Wilson, he studied the action of quinine sulphate on unicellular organisms primarily, and proved, as have many other investigators heretofore, that fairly strong solutions of quinine hydrochloride cause a distinct diminution in the activity of these organisms. It will be remembered that Binz more than thirty years ago showed that quinine distinctly prevented the migration of white blood cells during inflammatory action in a frog's mesentery, and similar results were obtained by the writer of this editorial and were published in a graduation thesis in 1884. In these early experiments it was found that the free diapedesis of leucocytes in an inflamed area was not only checked, but that the white cells which accumulated in large numbers in the capillaries of the affected parts failed to pass through the vessel walls. In Binz's studies the amount of quinine given varied from 0.01 to 0.005 per cent of the animal's body weight.

It is not necessary for us in this editorial to go deeply into the technique of the experiments which were carried out by Wilson. It suffices to state that he found, in addition to the effects already described by Binz and the writer, that the phagocytic power of the leucocytes was very markedly affected by the drug quinine. If large doses were administered it was inhibited, but if smaller doses were given it was very markedly increased, the increase amounting to as much as 25 per cent over normal phagocytosis. The quantity of quinine which produced this increase in phagocytosis was equivalent to about ½ grain circulating into the blood of a man weighing 170 pounds, or, in other words, that quantity of quinine which would probably be taken into the circulation from a dose of from 2 to 3 grains of the sulphate. It is a noteworthy fact that when the doses of quinine were very large, the drug was found not only exercising an inhibitory influence upon phagocytosis, but to be absolutely destructive. Wilson is unable from his experiments to state positively whether this increased ability of the phagocytes to destroy invading microörganisms depends upon the quinine rendering the bacteria very susceptible to the action of the leucocytes, or whether its effect is produced by increasing phagocytic activity; in other words, he has not determined whether the quinine increases the opsonic influence, or whether it has a direct action upon the white cells.

In the research of Manwaring and Ruh similar results were obtained, and as the two researches were entirely independent of one another, one can be considered as the strong indorsement of the other. Manwaring and Ruh not only found, as did Wilson, that quinine caused a stimulation in phagocytosis, but furthermore that large doses diminished phagocytosis, and if the dose was very large, arrested it entirely.

These two researches would seem to throw a flood of light upon certain empirical facts which have been known to the profession for many years. It has been held almost universally by active physicians and surgeons that quinine, whisky, and iron afford the best combination that we have for combating septic infection. If, as proved in the research already referred to, alcohol increases the bacteriolytic power of the serum, and if, as seems to

be proved by the two researches just published, quinine increases the phagocytic power of the blood, it becomes clear why quinine is so efficient a remedy in moderate quantities for combating septicemia, and, in the same way that the use of quinine in malarial fever was removed from an empirical to a scientific basis by the discovery that the drug destroys the parasite which causes this disease, we now have shown that the purely empirical use of this drug for other purposes seems to rest upon an equally sure scientific foundation.

In this connection, too, the facts asserted by these investigators in regard to the difference in effect produced by large and small doses are well worthy of thought. We have long been impressed with the idea that the principle that "if a little is good more is better" in the use of quinine is erroneous. Doses of 30 to 60 grains of this drug, which are sometimes administered in the conditions which we have named, are not only capable of producing manifest evidence of general systemic depression, but as these researches show are actually destructive to one of the chief activities of the body in protecting itself from disease; whereas small doses which are not sufficient to disorder digestion or to cause severe headache may produce excellent results.

In this connection, of course, the question as to the quantity of quinine which enters the blood is to be considered. Those persons who have studied most thoroughly the influence of quinine upon the malarial parasite seem to be convinced that if absorption is active very much smaller doses are required than those which many practitioners have been in the habit of administering. Thus, Thayer asserts that 15 grains given to a patient with malarial infection who is kept in bed at rest, and whose alimentary canal is active, is quite adequate to produce the necessary therapeutic effect. In many instances the condition of the stomach is such that even if 60 grains is given only a very small dose actually enters the blood. Physicians should recognize the importance of aiding in the actual absorption of the drug, remembering that the mere fact that medicine is in the stomach does not prove that it is active in producing its curative effects.

We trust that investigations such as those which we have just quoted may be continued still further, and that the use of a very large number of remedies, which are now employed as a result of practical experience without scientific indorsement, will be placed upon a firm and intelligent foundation.

THE LACK OF THERAPEUTIC VALUE OF CACTUS GRANDIFLORUS.

For a number of years a considerable number of practitioners have been under the impression that cactus grandiflorus possesses certain virtues as a cardiac stimulant, while others have considered that its stimulant effect is feeble, but have believed that it exercised a sedative or regulating influence upon the cardiac viscus. Thus, a well-known practitioner of Philadelphia has been accustomed to employ the tincture, or fluid extract, in cases of cardiac palpitation or irregularity, and while he has frequently combined this remedy with other drugs he has been wont to credit the good results to the cactus rather than to the remedy administered simultaneously. Those who have been most rational in this matter have, however, never believed that cactus possessed very great power, and certain investigations which have been carried on during the last few years seem to prove pretty clearly that even a moderate degree of activity is not possessed by this drug.

The most recent of these contributions is an investigation which has been published in the *Journal of the American Medical Association* of September 21, 1907, by Hatcher, who has studied the effects of the drug upon animals, and who has also gone over, in considerable detail, the literature which deals with this so-called remedy. After proving that the few experimental studies which have been made with it are not worthy of confidence, he proceeds

to detail the results which he has obtained in experiments upon animals, and he finds that cactus and its so-called "active principle" are not only devoid of any influence similar to that of digitalis and strychnine, but that they are inert when used on animals in doses that are hundreds and even thousands of times as large as those recommended by persons who claim that the drug produces excellent results. These conclusions distinctly indorse those reached by Sayre and Houghton which were published in the THERAPEUTIC GAZETTE in 1906. It is remembered that the good results which have been obtained from the use of cactus by certain practitioners may have been in reality due to other causes than the drug itself. The rest in bed which many practitioners wisely recommend to patients with cardiac irregularity is always a powerful factor in recovery, and the psychic influence of taking a supposed remedy from a physician in whom the patient has confidence does much toward diminishing mental anxiety in regard to the heart, particularly if at the same time some sedative like hyoscyamus, the bromides, or belladonna have been given as adjuvants.

THE USE OF TUBERCULIN FOR DIAGNOSTIC PURPOSES.

The editorial columns of the THERAPEUTIC GAZETTE have set themselves so distinctly in opposition to the employment of tuberculin for diagnostic purposes in pulmonary tuberculosis that it is only fair for them to occasionally present views which are in favor of this method of reaching a diagnosis of tubercular infection. It will be remembered that the reason for our opposition to the use of tuberculin originally consisted in the difficulty in obtaining a reliable product, and more important, in the idea that if a patient manifested a sufficient number of signs of tubercular infection to justify us in using tuberculin, then the evidences of tuberculosis were well enough established to make it wise to treat him as if he were a tubercular patient, since if actual tubercular infection had not occurred the patient was at least so fair a mark for such infection that every measure should be taken to improve his health and prevent further disease. Furthermore, it has not seemed justifiable to us, in a large proportion of cases at least, to subject a patient, on what might be called "the border-land of illness," to the reaction, or, in other words, the chill, fever, and sweat, which take place in most tubercular patients when tuberculin is given. There can be no doubt that the use of tuberculin, like many laboratory methods of diagnosis, very materially aids the physician in reaching a diagnostic conclusion, and in certain difficult cases in which tuberculosis is present in other tissues than the lungs its employment is undoubtedly justifiable. In pulmonary cases, however, in the vast majority of instances the careful development of physical signs will lead the physician to a diagnosis without this test.

In a paper in the *Boston Medical and Surgical Journal* of May 30, 1907, Hawes and Floyd point out that in the rush of a large out-patient clinic there is nothing harder than to make what might be called an early diagnosis of pulmonary tuberculosis; yet they appreciate the fact that if the disease is detected in an early stage it offers every chance of a cure or arrest. Under these circumstances, of course, the administration of tuberculin will aid a physician in reaching a diagnosis, but the question is whether a greater amount of time is not lost by the necessary preliminary and subsequent observation of the patient when tuberculin is given, and whether it is justifiable to subject the patient to this loss of time and to the reaction produced by tuberculin simply because there is a rush of work. It goes without saying that these authors are strongly in favor of its use as a diagnostic agent, and they quote with disapproval the statement of Knopf, of New York, that he "is not in favor of using it for the purpose of diagnosticating tuberculosis." They also quote Trudeau as saying that "any danger of aggravating the patient's condition lies in its faulty or irregular administration." There is a good deal in this statement of

Trudeau's. It seems to be pretty well decided by those who employ tuberculin most that it is not an agent which can be used indiscriminately by the general practitioner. On the contrary the infinitesimal doses which should be used at first and the observation of the results which follow seem to be confined to those who may be considered experts, and Dr. Frank Billings very sensibly takes the ground that the use of tuberculih may be dangerous by individuals who do not use other methods of diagnosis which would ordinarily be sufficient. It goes without saying that tuberculin ought not to be used in those cases which are manifestly tubercular, first, because the reaction is disagreeable to the patient, and secondly, because the injection is absolutely unnecessary. Furthermore, it should not be used in cases which have recently suffered from hemorrhage and which have definite signs in the lungs, or which are already febrile. In bone disease and in abdominal tuberculosis it is without doubt a valuable diagnostic agent.

As the result of employing tuberculin in a very considerable number of cases Hawes and Floyd conclude that tuberculin is an agent of very great value in early tuberculosis; that it should be used only as a last resort after all other means of making a diagnosis are exhausted; that it should be used only in carefully selected cases by men properly trained and qualified to employ it; that the initial dose should not be over .1 of a milligramme; that it is best to watch such patients in bed even if they may be "walking cases"; and finally, that if a patient reacts to tuberculin and at the same time has a history and physical signs which leads one to suspect tuberculosis, he should be treated as a tubercular case. It seems to us that these very sensible conclusions meet the indications for the use of tuberculin, and perhaps the most important of them is the second one, namely, that it should be used only as a last resort after all other means of making a diagnosis are exhausted.

In this connection the conclusions reached by Rotch and Floyd, and published in the *Journal of the American Medical Association* of August 24, 1907, are of interest. These investigators studied the relative value of the opsonic indexes and the tuberculin test in the diagnosis of early tuberculosis in children. They conclude that tuberculin is a valuable diagnostic agent in early tuberculosis, but when not used properly is dangerous, and in inexperienced hands may bring disaster, and they regard reaction in children as consisting not only in a rise of one or more degrees of temperature, but in the development within twenty-four to thirty-six hours of headache, nausea, vomiting, and diarrhea, with marked tenderness in the seat of the injection, and the appearance of cog-wheel or harsh breathing, and râles over the suspected area. Indeed, they consider the development of these pulmonary symptoms during a reaction as being of great importance.

We again reiterate what we have said before, that in our experience there are few cases of pulmonary tuberculosis in which the use of tuberculin as a diagnostic agent is essential, and it seems to us that this is particularly true in children, in whom pulmonary tuberculosis is comparatively rare, in whom the development of a reaction scarcely seems justifiable, and in whom the evidences of tuberculosis are usually sufficiently well developed to make it wise to treat the patient as one so affected, even if the diagnosis is not scientifically positive.

SOME PLAIN TRUTHS ABOUT SYPHILIS.

Bulkley, speaking of the extent to which syphilis prevails, holds that the best criterion in regard to this point is to be found in the practice of the dermatologist, since the disease in some portion of its evolution infallibly affects the skin (*New York Medical Record,* vol. lxxii, No. 6, 1907). He notes that of several hundred thousand cases of miscellaneous diseases of the skin compiled from the practice of members of the American Dermatological Association, from various cities all over the

United States, syphilis figures as the causative agent in about 10 per cent. In his own published statistics of 20,000 personal dermatological cases, in public and private practice, syphilis formed over 12 per cent of the number. Arguing from this fact alone it is fair to infer that syphilis is a comparatively infrequent disease, since the total number of patients suffering from any skin lesion of sufficient severity to require a specialist forms a very small fractional percentage of the total population. It is, however, true that the great majority of syphilitic patients suffering from skin lesions do not consult the dermatologist, but are treated by their family practitioners. Therefore these figures are of little use in forming a basis for the calculation of the total instance of syphilis.

Bulkley's second truth is that syphilis is not necessarily a venereal disease. He quotes Fournier to the effect that nearly 25 per cent of females contract syphilis innocently and undeservedly, whilst among married females the disease is unmistakably traced to the husband in 75 per cent of all the cases. These statistics are corroborated by Ricord. Bulkley states that in his private practice a careful study and analysis of his cases reveal that in somewhere near 50 per cent the disease is acquired in an innocent manner wholly apart from sexual transgression. With this estimate most men of experience will agree, though perhaps each will give a different percentage. The fact certainly remains that a large number of people contract syphilis in ways other than by sexual congress.

In regard to the contagiousness of syphilis Bulkley marvels, as do many others, at the existence of a false shame which absolutely prevents any far-reaching public efforts in the direction of prevention.

To the seriousness of syphilis Bulkley accords a just importance.

As to its tractability, this he holds is dependent upon a full and perfect treatment continued until the poisons are eliminated from the system. The two-year limit of treatment he seems to regard as adequate. He cautions against alcohol, even in moderation, holding that an efficient treatment depends upon total abstention. Toward tobacco he is more tolerant, though he believes it had best be avoided.

Mercury, he states, is still the one and only remedy which exercises a specific control over the disease. It should be so given that it permeates the system with the least harm. Bulkley himself usually administers mercury by the mouth up to the point of tolerance. The hypodermic administration of mercury he regards as a valuable addition to our means of combating syphilis in many cases, but does not favor it as a routine method of treatment.

The plain truth in regard to iodide of potassium is that in itself this drug has really no effect upon the syphilitic poison, though it is often erroneously given for this purpose, but as an adjunct to mercury for some of the later results of syphilis it is of inestimable value, and it probably aids materially in effecting the rapid elimination of mercury from the system. Bulkley particularly cautions against the common error of employing potassium iodide alone in the later stage of the disease, and holds, as do nearly all who have had large experience, that more or less mercury is needed as long as there are any manifestations of the disease. He observes that the enormous doses of iodide of potassium which are sometimes given are commonly both useless and irrational.

In regard to the supposed value of mineral springs in connection with syphilis Bulkley certainly voices the opinion of the majority of those most expert when he states that much harm often results from the false security which patients have who have undergone what is called a cure at one of these places. It has been clearly demonstrated that syphilis cannot be cured by any brief course of treatment, however severe. It is true that the cures are distinctly beneficial. This is almost unquestionably due to the active and heroic mercuric treatment to which patients have been subjected combined with change of air and scene, together with the natural hope and expectation of benefit. Bulkley does not recall a single instance in which he has regarded it as either beneficial or

wise for the patient to undertake this cure at any great personal sacrifice.

In this somewhat informal paper Bulkley so well epitomizes the beliefs and practices of those most skilled in the treatment of syphilis that his remarks are worthy of careful consideration. Perhaps the most important truths to which he draws attention are the efficacy of mercury in all stages of the disease and the vital importance of attention to the general health.

THE HYPEREMIA TREATMENT OF SURGICAL TUBERCULOSIS.

In spite of the now very large amount of clinical trial accorded to the Bier treatment by passive hyperemia for the relief of all forms of local infection, it cannot be said that in this country the treatment has received a wide acceptance, nor that it has been attended by uniformly satisfactory results.

It should be accepted as a fact that any form of treatment enthusiastically advocated by an able man who believes in it will not only be practiced by others, but that brilliant, often extraordinary, results will be reported as a consequence of this practice. It is not till results are usually successful in the hands of the majority of practitioners that a given treatment can be regarded as having established its value. In accordance with this standard Bier's treatment must be recognized as still in its experimental stage. This well may be, not because it is wanting in merit, but because it is too often employed by those who lack judgment either in its method of application or in the selection of cases likely to be helped by its use.

Bier's basal principle is the direction of treatment against infection, not against inflammation, which he regards—and indeed so do we all—as desirable, helpful, and in itself protective against the infection. He holds that if he employs such means as increase local inflammatory symptoms rather than diminish them, he is thereby aiding the body tissues in their struggle against the germs.

Thus he is diametrically opposed in his fundamental principles to those who believe that by the elevation of the part, by the application of evaporating lotions, by efforts designed to lessen swelling, heat, and redness, cure is most promptly and efficiently accomplished. The heat, redness, and swelling Bier believes are the indices of natural reaction, and means should be taken to increase them rather than diminish them.

The practical application of Bier's theory lies in increasing the amount of blood in the inflamed area and in retarding the activity of the circulation. This is accomplished by constricting bands so placed as to interfere with venous return.

Schmieden (*Medical Record*, vol. lxxii, No. 7), who has acted as Bier's assistant in the elaboration of the technique, notes that tuberculosis, especially in its joint manifestations, was the first disease which Bier treated with his hyperemia band. There was some difficulty at first in arriving at a correct judgment as to the proper degree of hyperemia and the length of time during which this should be maintained.

Schmieden's description of the technique is as follows: A 2½-inch rubber band is applied slowly and evenly around the extremity, proximally but not too close to the tuberculous joint. Each turn of the bandage overlaps its predecessor. It must be applied so gently that no pain is caused either at the place of application or in the diseased joint. This is regarded as a matter of cardinal importance. When properly applied the extremity below the bandage begins to swell and assume a bluish-red color. It should remain at least as warm as the other limb, and the pulse should remain entirely unchanged. There is thus produced not a stoppage of the blood current, but an increase in volume due to a slight obstruction to the return flow.

In tuberculosis the hyperemia bandage is applied only a few hours a day—one to four hours as a rule, the time being so divided that the band is put on once for an hour or once for two hours, or twice for an hour or twice for two hours. When

the skin is very tender the location of the band is changed.

During the hyperemic treatment all local dressings are removed, and a piece of sterile gauze or towel is placed over the discharging fistula, since a pressure bandage of any sort makes the hyperemic treatment impossible.

Nor should the joint be kept in fixation. When the articulations of the upper extremity are involved the patients are allowed to use a sling for support, and soon are permitted to make slight exercises with the arm, as, for instance, in dressing themselves.

In severe tuberculosis of the knee absolute rest in bed is imperative. With improvement the patient should be gotten up, the joint being protected by a mechanical apparatus which whilst allowing movement prevents pressure. Bier does not regard a stiff joint as a good result, but holds that resection of a joint is, in regard to cure, an illusion. Though he admits that it may cure the tuberculosis, it mutilates by destroying the joint.

The first sign of betterment is the abeyance of pain, which is in itself a certain proof of proper technique. This is followed by disappearance of inflammatory contracture, making active and passive movements possible. In the course of treatment swelling subsides, and large fungous masses are transformed into connective tissue, which later by hyperemic treatment are absorbed. Gradually the contour of the bones again becomes distinctly visible. Tuberculous fistulæ, even though they extend into the joint or bone, close.

Often cold abscesses develop. Bier's treatment for these is by opening and the application of cupping-glasses. Indeed, the formation of a cold abscess indicates a process by means of which the organism casts off the tissue incapable of healing. Its development constitutes no contraindication to the continuance of the congestive treatment.

As to the time required for this treatment, Schmieden states that the patient who desires to be healed quickly must submit to resection or amputation, but that nine months, or even longer, is necessary for the cure of a severe affection of a joint.

Schmieden is evidently an enthusiastic advocate of Bier's principles and practices, and regards as especially in favor of the latter the circumstance that the treatment is so simple that it can very shortly be applied by the patient himself or by any one who is in the house with him. Every ordinarily intelligent patient soon learns to find when the band is properly applied. Experience in a large clinic has shown, according to the author, that this treatment is applicable to nearly all cases. The hip-joint is, however, still beyond reach. The results are particularly favorable in tuberculosis of the ankle, elbow, and wrist. Even such cases as were formerly resected apparently yield readily to treatment, nor is the severity of the disease ever a contraindication to hyperemia.

The treatment is just as effective in the old as it is in the young. As the result of success attending this treatment Schmieden notes that very few resections are done in Bier's clinic, the resections being confined to patients whose general condition is very bad, in whom amyloid degeneration is present, or there is wide-spread tuberculosis of the lungs.

The knee-joint requires special treatment: in the early stage of the disease extension and the application of splints during the time the patient is not in bed, thus supplementing the hyperemia. Those cases in which subluxation of the leg has occurred, or ankylosis between the patella and femur, are better and more quickly treated by resection, since there is then no hope of a movable joint.

The exudative form, so-called hydrops, is never treated by hyperemia. It is aspirated and injected with iodoform emulsion. All cases are radiographed, and even though a bone focus is found its presence in no way contraindicates the treatment by hyperemia.

In regard to the use of the cupping-glasses, these are indicated in all cases of beginning softening and of abscesses which may or may not have fistulous openings. The cups are allowed to remain in place

three-quarters of an hour, but with recesses of about three minutes every five minutes.

The hyperemia must not be too intense. The skin should be red, not blue. The glasses will stay in place if their edges are coated with vaselin. All tuberculous abscesses are opened promptly without fear of mixed infection, since the proper application of cupping glasses will always prevent this. There is evacuated daily through a fistula all detritus, however deeply it may be situated. The pus and detritus having been evacuated, the discharge becomes serous, steadily decreasing, until finally it ceases. Tamponade and drainage are unnecessary, indeed are hurtful, nor is there any other treatment so agreeable to the patient. In all cases of open tuberculosis of joints a hyperemic band is applied daily, twice for an hour, and the cupping-glasses over the fistula for forty-five minutes.

To those familiar with Bier's writing and teaching, particularly his book on "Hyperemia as a Therapeutic Agency," the brief summary of his views and of his practice in regard to the treatment of joint tuberculosis will be without help. To the vast majority, who have not ready access to current literature, or who are so busy earning their daily bread that they have no time for reading, such summarizing of principles as is given in Schmieden's communication is peculiarly valuable. This particular paper shows to any one conversant with hospital services and current literature that the Bier treatment has often been taken up by those totally unfamiliar with either its principles or application, and that the failure resulting therefrom may be justly attributed to this ignorance.

Making due allowance for the enthusiasm of the scientist for his creation, Bier's enunciation in the fifth edition of his book, that "he formerly considered the hyperemic treatment of tuberculosis good, but now considers it the best," should carry considerable weight.

REPORTS ON THERAPEUTIC PROGRESS.

ENURESIS AND FECAL INCONTINENCE IN CHILDREN.

STILL says in the *Clinical Journal* of April 24, 1907, that before deciding on the mode of treatment the urine will have been examined, and his experience has been that, although occasionally some abnormality may be found in the urine, in the vast majority of cases the urine is perfectly normal and gives no guidance whatever. Occasionally a concentrated acid urine may call for the use of potassium citrate; the presence of uric acid crystals may be met by cutting down the carbohydrates as far as practicable—a measure which he believes to be more useful in such cases than cutting off meat and other nitrogenous food, as is usually done; or again, the presence of pus or bacteria in the urine may indicate the need for urotropin or uritone, which can be given to a child of about five years in doses of 4 to 5 grains three times a day.

Far more often an important indication for treatment is found in the presence of thread-worms, and it is often wise even when their presence is denied to give one dose of santonin 1 to 2 grains with calomel 1 to 1½ grains, according to the age, to make this point certain, for there is little chance of stopping enuresis whilst worms are present.

In the majority of cases, however, no exciting cause offers itself for treatment, and then nothing is more generally useful than the time-honored belladonna. But to obtain the best results from belladonna it must be given on a definite plan, and for a period of several weeks. The random bottle of belladonna is responsible for much discredit in the treatment of enuresis. The parents should be given to

understand at the outset that the drug is a potent one, and that the child must be seen at intervals of a few days by the doctor in order that the dosage may be carefully increased as the child is able to tolerate it. If this is clearly understood there is some hope of doing good with belladonna.

Many failures are due to the use of doses which are far too small. The writer has seen cases given up as incurable in which no more than 5 minims of the tincture three times a day had been tried. Children usually tolerate belladonna well, and at any period beyond infancy 5 minims of the tincture should only be considered as an initial dose. If the parents are intelligent and are forewarned of the possible toxic symptoms, it is usually safe to begin with 10 minims for a child over five years of age. The dose should be increased by 2½ minims every fifth or sixth day until either the enuresis is stopped or the limit of tolerance is reached.

If the enuresis is controlled, say, by 17 minims three times a day, it is well to "make assurance doubly sure" by pushing the dose slightly further by an additional 2 minims if tolerance allows. The dose should be maintained at this level, just beyond the minimum efficient dose, for a fortnight, and then reduced by decreases of 2 minims once a week, and so gradually discontinued.

A word of caution with regard to belladonna: children, like adults, have their idiosyncrasies to drugs, and while some will tolerate 30 minims of the tincture of belladonna three times a day, the writer has occasionally met with marked intolerance even of so small a dose as 5 minims. Another caution which applies especially to such a drug is this: see that the dose is measured with a medicine glass, not with a spoon; the domestic teaspoon will often hold two drachms, sometimes as much as three drachms. Nor is even a medicine glass necessarily reliable if it be one of the cheap measures which are often used. The author found in one case that a drachm measured by a cheap medicine glass was actually two drachms, and smaller errors were found to be quite common.

There are many cases in which, after the belladonna has been pushed up to 17 minims or 20 minims with decided diminution of the enuresis, the disorder is still not completely controlled, and yet any further increase of the belladonna produces toxic symptoms which are sufficient at any rate to alarm the parents. Under these circumstances a useful procedure is to keep the belladonna at the topmost dose which is well tolerated, and to combine with it tincture of lycopodium, 12 minims at first, with increases of 2 minims as may be required. A child will usually take 20 minims of this drug without ill effect, sometimes more, and certainly in some cases with most gratifying result.

DISEASES OF THE NASOPHARYNX IN INFANCY.

In writing on this subject in the *Boston Medical and Surgical Journal* of April 18, 1907, MORSE reminds us that adenoids are one of the commonest causes, if not the most common cause, of chronic "snuffles" in infancy. They are also almost always present in those babies that are subject to frequent "colds in the head," and are of great etiological importance in this connection. They are usually overlooked, however, because the baby does not keep its mouth open, snore at night, or have the typical facies of adenoids in later childhood, there being apparently a very general impression that there can be no adenoids, or at any rate no adenoids of importance, unless these symptoms are present. This is not so, of course; frequent colds and chronic snuffles are almost as suggestive and characteristic of adenoids in infancy as those more marked symptoms are in childhood. The treatment of these affections is most unsatisfactory, moreover, unless the underlying condition is recognized. When this is done and the adenoids removed, the "colds" and "snuffles" cease almost immediately. Opera-

tion is not always necessary, however, as mild cases often yield to local astringent and stimulating treatment. The author has found the following mixture very useful:

Iodine, ¼ to ½ grain;
Camphor, 1 grain;
Menthol, 1 grain;
Benzoinol, 1 ounce.

Five to ten drops of this mixture are put into the nose with a dropper every three or four hours. This, by the way, is the best method of making applications to the nose and nasopharynx in infancy. Sprays are of little use at this age because of the fright and struggling which they induce. The drops are best put in with the baby lying on its back so that they may run through into the nasopharynx. Although these cases are so common and the relief from proper treatment, especially from operation, is so striking, it hardly seems necessary to give illustrative cases, as the condition is easily recognized if it is only borne in mind.

Adenoids not only cause "snuffles" and "colds," but also very frequently an irritating cough without physical signs. This cough is especially troublesome at night. Here again treatment, unless the real source of the trouble is recognized, is of no avail. Cough mixtures and throat sprays are useless. The cough can only be controlled by stupefying the infant with bromide or paregoric. Treatment of the adenoids or their removal, however, is almost immediately successful.

THE TREATMENT OF MALARIAL FEVER.

VANDERHOOF, in the *Journal of the American Medical Association* of April 20, 1907, asserts that in the treatment of the individual case emphasis must be laid on the following points: (1) The quinine must be absorbed and enter the blood. (2) The drug must be administered until every parasite is destroyed. (3) The patient should remain in bed until the temperature reaches normal and remains normal.

In order that the quinine may be absorbed there are two essential features to which attention must be directed: (1) The patient's alimentary tract should be in a condition to favor absorption. To this end the bowels must be unloaded, followed by a saline. The diet should consist of soft, easily digested foodstuffs, and as long as there is fever liquid diet alone is preferable. (2) The quinine must be given in a soluble form. In this connection the writer cites briefly a rather remarkable case that came under his notice a couple of years ago. A man was brought into the hospital in a state of collapse whose blood presented an extreme picture of pernicious malaria, nearly every other red cell containing one or more parasites. He died in a few hours in spite of heroic treatment, and at the autopsy was found an ischiorectal abscess which contained in its pocket-like recess a very large number of quinine pills which had passed through the intestine without being at all dissolved. Such an experience as this is certainly striking enough to condemn forever the administration of quinine in pill form. Undoubtedly the surest way to give quinine is in solution with a mineral acid—*e.g.*, quinine sulphate 5 grains, dilute hydrochloric acid 5 minims, water to 1 drachm. It is not very palatable, although the acid seems to offset somewhat the bitter quality of the quinine, and the writer has always found that patients take it without objection. The drug may be administered in powdered form in soft gelatin capsules, but it is not an uncommon experience to have them pass entire from the rectum. The solubility of the quinine when given in this form is aided by following the dose in a few minutes by 10 minims of dilute mineral acid. The best indication we have that the quinine is absorbed is the subjective sensation of ringing in the ears, and if this is lacking we may feel quite sure that the quinine is not in the blood, except possibly in certain unusual cases of idiosyncrasy in this regard.

The necessity of giving quinine for a definite period after the fever and symp-

toms have disappeared is not always sufficiently appreciated. Last fall the writer had several patients brought to him who gave a history of chills and fever five or six weeks previously, who had been given quinine for a few days, which caused the symptoms to abate, but they have never felt quite well since, complaining chiefly of periodic headache, malaise, and occasional chilly sensations, and on examination the blood still showed the plasmodium. A good general procedure in the handling of ordinary cases is the following: 5 grains of quinine every four hours, or 20 grains a day, for the first five days; then 2½ grains every four hours, or 3 grains three times a day, for two and a half or three weeks. In severe cases larger amounts may be given, 30 to 40 grains daily. It is generally advisable to give one large dose, 10 or 15 grains, five hours before an expected paroxysm, and if this is done there is usually sufficient quinine in the blood to destroy most of the young hyaline forms that are produced by segmentation during the chill. But if a case is seen directly after the rigor and another is not expected for two days the author much prefers to start the quinine at once, as it is undoubtedly true that a certain number of parasites are destroyed by the quinine while still contained within the red corpuscles, and the succeeding paroxysm is much modified. Let it here be again emphasized that quinine is a specific in malaria, that if it enters the blood in sufficient quantity the fever should disappear in a few days, certainly within a week, and the writer doubts very much whether it is possible under these conditions for malarial fever to run as long as thirty days, a remark that is sometimes heard. Dr. Carter in a communication touches directly on this point, as in over 200 cases of estivo-autumnal fever treated in the canal zone at Panama the average duration of fever was only fifty-two hours in this severe type of infection.

In pernicious malaria every means should be employed to get the quinine into the circulation as quickly as possible. The drug must be given hypodermically—or, rather, intramuscularly—or even intravenously, and may also be given by the rectum. Tyson objects to the hypodermic method, stating that he has had to abandon it on account of the frequency with which it is followed by abscess formation. Undoubtedly quinine is very irritating to the tissues, but if the precaution is taken of injecting it deeply into the muscles and not simply under the skin, abscess formation is less often encountered—at least this has been the experience in Panama, where this method is frequently used. The bimuriate of quinine, or the bisulphate of quinine and urea, are the two preparations most frequently used. These methods of administering the drug are dealt with in most text-books on the subject.

The necessity for absolute rest in bed while actively combating the infection must be apparent to any one who has tried the simple experiment of treating an ordinary case of tertian malaria by this means alone without the use of quinine at all. While by no means the best way to kill off the parasites, yet it is surprising to see what nature can do when aided by rest, diet, care of the bowels, and general hygienic measures. It is very certain, however, that the rest plus the quinine is more efficacious than either alone.

Appropriate means should be employed during the paroxysms to make the patient as comfortable as possible—blankets, hot-water bottles, and possibly a hot whisky-and-water drink. The judicious use of morphine is often indicated, especially if there is much gastric disturbance. A tepid sponge or an alcohol rub is appreciated during the sweating stage.

The postmalarial anemia is best treated with some form of arsenic. The author usually prefers Fowler's solution, running up to 10 or 12 minims three times a day, provided the patient takes it well. Some prescribe a combination of iron and arsenic in pill form. If there is any indication to continue quinine, such a tonic as the syrup of iron, quinine, and strychnine phosphate may be given.

THE PHARMACOLOGY OF ETHYL CHLORIDE.

Dr. E. H. EMBLEY, honorary anesthetist to the Melbourne Hospital, has contributed to the Royal Society the results of an experimental study of the pharmacology of ethyl chloride with the view of saving what appears to him to be a valuable anesthetic agent from coming under the suspicion of being unduly dangerous. These results arrive at an opportune moment, when the merits of ethyl chloride are so much under discussion. For comparison with the effects of chloroform Dr. Embley used dogs in his experiments, and employed morphine and ether as the preliminary anesthetics. The solubility of ethyl chloride vapor in water at normal pressure and 21° C. was found to be 253.36 per cent by volume (0.678 per cent by weight); its solubility in blood was found to be approximately 500 per cent by volume of the vapor at 38° C.

Dr. Embley next determined the effect of ethyl chloride upon the heart isolated from the central nervous system by a special method of ligation of the arteries, by means of which a circuit was maintained without the introduction of cannulæ. It was found that the heart muscle is paralyzed by ethyl chloride, as by chloroform and in contrast with ether, but the quantity of ethyl chloride vapor in the air required is nineteen times as great as that of chloroform to produce similar results.

The effect of ethyl chloride upon the vascular system was then investigated. The net result is dilatation, but the degree of paralysis is strikingly less than that produced by chloroform, even when the latter is present in less than one-tenth the quantity in the air inspired.

From a study of the effect of ethyl chloride upon the vagus mechanism Dr. Embley concludes that vagus inhibition of the heart occurs very readily when ethyl chloride vapor of a strength of 10 per cent and upward in the air inspired is administered. A sudden fall of blood-pressure occurring when the air inspired contains from 10 to 20 per cent of ethyl chloride is due to vagus inhibition of the heart. When the strength is increased to 30 per cent and upward the fall of pressure is due to weakening of the cardiac and arterial musculature. Dr. Embley is of opinion that cardiac inhibition is not so serious from ethyl chloride as it is from chloroform, since it comes on before the spontaneous excitability of the heart has been much depressed. It does not seem possible permanently to arrest a dog's sound heart under ethyl chloride narcosis by vagus inhibition. It requires nineteen times more ethyl chloride to produce a given degree of cardiac depression than is required of chloroform, whilst it requires only four times as much to produce cardiac arrest by vagal stimulation, hence inhibition sets in relatively rapidly. Dr. Embley considers that these facts explain the relative safety of ethyl chloride. It was also observed that the cardiac inhibition is not a reflex but arises from central stimulation. There was no evidence of any paralysis of vagus endings such as was described by Cole. Dr. Embley further shows that in ethyl chloride narcosis the integrity of the respiratory mechanism is dependent upon the maintenance of blood-pressure, as in the case of chloroform. Paralysis of respiration may be preceded by shallow respiratory movements, but it is usually preceded by deep and delayed breaths, and in some cases the prolonged pause prevents the fall of blood-pressure. In all the experiments the respiration failed before the heart. A strength of from 5 to 7 per cent of ethyl chloride vapor in the air respired appears to be the limit of safety from danger of syncope in dogs for prolonged and continuous administration. If this concentration be exceeded it is inadvisable to continue the administration beyond abolition of the corneal reflex, as otherwise syncope is very probable.

Dr. Embley considers that these conclusions with regard to the effects of ethyl chloride upon dogs apply to man. He has observed the occurrence of syncope in man in two cases, in each of which recovery took place suddenly. From analogy with experimental results he regards such syncope as due to vagus inhibition, except in cases of failing heart and other morbid states. A

sound heart is able to free itself from vagus inhibition, but the degree of depression caused by ethyl chloride may be sufficient during the period of syncope to embarrass an unsound heart beyond recovery.

As regards the administration of ethyl chloride, Dr. Embley advises the employment of a gasometer containing the mixture of air and ethyl chloride vapor. He considers that less safety is assured by the use of any apparatus by which the anesthetic is sprayed or poured into the instrument between the face-piece and the bag, whereby during inspiration excessively high degrees of concentration of ethyl chloride may be given. It is better, he asserts, to mix the anesthetic with the air at the end of the bag.—*Lancet*, April 20, 1907.

ON THE USE OF A NEW FLUID FOR THE HEMATOCYTOMETER.

To the *Lancet* of July 13, 1907, EDINGTON contributes a paper in which he states that of all the fluids in use—and there are a considerable number—the best that can be said of them is that they make an approach to being isotonic with normal blood. "A solution containing just a percentage of salt as suffices to keep the corpuscles unaltered in form and without removal of any of their hemoglobin is 'isotonic.' For human blood a solution of common salt is isotonic with 0.9, for defibrinated ox blood with 0.6, and about the same for frogs' blood. Very slight differences of external condition will tend to alter the permeability of the blood-corpuscles both for hemoglobin and for other substances" (Schäfer's "Text-book of Physiology").

The normal specific gravity of the blood in a healthy subject is from 1058 to 1062, but in departures from the normal the specific gravity may be much lower, and has been found even so low as 1033. With a lower gravity the hemoglobin falls in an almost constant fashion, so that while with a specific gravity of 1057-60 there may be 85 to 95 per cent of hemoglobin, with a specific gravity of 1033-35 hemoglobin will have fallen to 25 to 30 per cent. When coagulation is prevented the corpuscles of drawn blood very soon sink, leaving the yellow plasma as a supernatant fluid, and it is evident that if hemoglobin is given up by the corpuscles to the plasma it is very soon eliminated from that fluid, most probably by the kidneys. In various kinds of experimental hemolyses the author has found free hemoglobin in the plasma antecedent to its appearance in the urine. It is probable, therefore, that changes in the amount of hemoglobin are in great part due to abnormal permeability of the corpuscles and also to their destruction. In any case, supposing the blood to be examined to be that of an abnormal individual, we may assume that the specific gravity is almost certainly below the normal. If this be the case, then the use of a fluid which is isotonic for healthy blood is almost certainly improperly constituted for a blood of lower specific gravity. It seemed to the writer, therefore, of some importance to determine whether one could not prepare a fluid which, without having great regard to the isotonic quality, would be useful for any kind of blood.

Some ten years ago he first used neutral citrate of potassium as a means to maintain large quantities of pathological bloods in a fluid condition so that they could be used for inoculation, and the plan he then adopted has, since that date, found its way into most bacteriological institutions. What, however, struck him as remarkable was the fact that the red blood-corpuscles maintained their shapes and characters in a marvelous manner, so that even blood films could be made with blood so prepared. He has therefore prepared the following solution for use with the hematocytometer: neutral citrate of sodium, 7.5 grammes; formalin (40-per-cent), 2.0 cubic centimeters; dahlia (Grübler's), 0.03 gramme; chloroform, 5 drops; and distilled water, 250 cubic centimeters. In preparing the solution it is better to mix the stain with the water, rubbing down the former with a few drops of the latter first, then to add the citrate and formalin. The solution should be left for a day or two

without filtering, so as to deposit all the less soluble portions of the dye. The solution is easily made and keeps well when kept in a dark and cool place. If, however, it has not been used for some time it is advisable to filter off a small portion for immediate use. In less than a minute after the diluted blood has been placed on the slide one will find the red corpuscles deposited on the glass so completely that nearly all are in focus, while the refractive index of the corpuscles is so well maintained that they are very easily counted.

In practice the author prefers to eject one or two drops from the tube of the hematocytometer, and thereafter to empty the remainder into a small glass tube which is closed by a well-fitting stopper. By this means the mixed blood and fluid may be sent long distances for examination. Messrs. Thomson, Skinner & Hamilton, of Glasgow, now supply such tubes at a nominal cost. The hematocytometer tube, after using this solution, is very readily cleaned, a circumstance which will commend itself to many. The rubber tube supplied with the Thoma-Zeiss hematocytometer is too short. The author prefers using a rubber tube of twice the length. He has also found a rubber ear syringe (one in which the ball and nozzle are composed of black rubber) to be most useful for washing the glass tubes, since one can affix the compressed ball to the tube and allow it to suck up water or alcohol for cleaning, and thereafter by compressing it the fluid is entirely discharged.

TRANSFUSION IN SEVERE ANEMIA.

Transfusion of blood in the treatment of severe anemia appears to be almost entirely given up, although some fifteen or twenty years ago it was freely made use of. Many older authorities have attempted to support the method, but the majority of the modern writers raise a number of objections to it. One must realize at once that transfusion is quite incapable of doing good in cases of tumor or of aplasia of the medulla bones, and in those processes in which there is a rapid and progressive destruction of red corpuscles. P. Morawitz (*Münchener med. Wochenschrift,* April 16, 1907) says that, apart from these cases, there is a long series in which the anemia depends, in part at all events, on the deficient regenerative power of the bone-marrow, but in which this power can be induced to recover itself. Systemic treatment by arsenic produces improvement and cure in this way. The objections to transfusion include the fear of ferment intoxication. The appearance of dyspneic attacks, edema, and fever were supposed to depend on an intoxication with fibrin ferment and a secondary capillary thrombosis. This, however, has proved to be erroneous, and it is now certain that intravascular thrombosis can only be produced by defibrinated blood when the blood is injected too fresh or too warm, or when the fibrin coagulum has been squeezed. It is impossible to prophesy when transfusion will be tolerated without reaction, or when rigors, hemoglobinuria, etc., will follow; but while these symptoms are sufficient to prevent the method from becoming popular, the author insists that no case of death has yet been recorded, and that it is just the cases in which the symptoms were well marked that the greatest good has been done. It must further be remembered that the cases for which transfusion may be required are cases for which one can do nothing in any other way. In France the transfusion method has been entirely replaced by treatment with hemolytic serum and organic extracts. These methods, however, are too new to justify one in giving up the old procedure, provided that the old method is capable of doing what is required of it.

In order to illustrate what transfusion is capable of doing, the author recites six cases in which he has carried it out. The method adopted was as follows: A healthy person, or one who had only some insignificant ailment, was chosen to give up the necessary amount of blood. The blood was obtained by puncturing the median vein after constricting the arm, and allowing the blood to flow directly into a sterile

glass bottle with a ground-glass stopper. The defibrination was carried out by shaking the bottle, which contained glass beads, for at least five to ten minutes after the blood had been drawn off. After standing for from twenty to thirty minutes the blood was filtered through several layers of sterile canvas into a second glass bottle, but the clot was not pressed. (If one withdraws about 250 cubic centimeters of blood, one will have about 200 cubic centimeters after it is defibrinated and filtered.) A cannula was then introduced into the median vein of the anemic patient by means of a small incision, and when the patient's blood had filled the cannula, a funnel with tube was attached to the same and the warmed blood poured into the funnel. The blood should not be allowed to flow quickly into the patient's vein, and the author found that if one allowed from twenty to thirty minutes for the whole 200 cubic centimeters one would not meet with difficulty. The cannula was then removed and the wound closed and covered. In the first case the patient was suffering from very severe anemia, and the characteristics in the blood count were like those of leukemia. Edema, ascites, and enlargement of the liver, loss of appetite, vomiting, and increasing illness persisted, in spite of arsenic and other forms of treatment. The transfusion was then carried out, and resulted in a steady recovery, the leucocytes regaining their original normal condition.

The second case was one of postpartum pernicious anemia. The symptoms here were extremely severe also, and were progressive; 150 cubic centimeters of defibrinated blood were transfused, and no reaction followed. From this time onward the patient made a good recovery.

The third case was another of pernicious anemia. Here, again, after other means had failed to check the progress of the disease, transfusion was carried out, and from that time improvement set in and continued until recovery was complete. The fourth and fifth cases were similar. A reaction was seen in the third and fifth cases. The recovery of the fourth and fifth cases could not be followed out completely.

In the sixth case the patient was suffering from a severe aplastic anemia. Here the transfusion, which was twice performed, failed to do any good, and death followed as the patient became more and more anemic. The post-mortem examination revealed atrophy or aplasia of the medulla of the bones. In this case no reaction followed the transfusion. The beneficial action of the transfusion was best marked in the first three cases, but in none did it set in immediately after the transfusion. A few days elapsed before the blood showed a disposition to improve. The immediate results in the fourth and fifth cases were somewhat alarming, but the untoward symptoms passed off safely.

The author discusses the possible action of transfusion from a theoretical standpoint, and concludes by stating that the method should be again revived for the treatment of severe forms of anemia.—*British Medical Journal,* July 20, 1907.

CAN ANTITOXIN, GIVEN BEFORE INFECTION AS A PROPHYLACTIC, PREVENT POSTDIPHTHERIC PARALYSIS?

ROSENAU and ANDERSON in Bulletin No. 38 of the U. S. Hygienic Laboratory answer this question as follows:

Postdiphtheric paralysis in the guinea-pig is an almost exact counterpart of the same sequel in man. We are therefore able to bring forward experimental evidence showing the effect of antitoxin upon postdiphtheric paralysis.

In the guinea-pig antitoxin cannot influence the diphtheric paralysis after the paralysis has appeared.

Antitoxin has no influence in preventing postdiphtheric paralysis if injected shortly before the paralysis develops.

Antitoxin given twenty-four hours after the infection can save the life of the guinea-pig and greatly modify the paralysis.

Antitoxin given in a single large dose

forty-eight hours after the infection did not modify the paralysis or save life. Thus, in their experiments 4000 units failed to modify the paralysis or save the life of guinea-pigs weighing about half a pound. Weight for weight this corresponds to 400,000 units for a 50-pound child.

Antitoxin given in repeated injections, beginning twenty-four or forty-eight hours following infection, seems to have a more favorable effect upon the subsequent paralysis than a single injection.

A very small quantity (1 unit) of antitoxin given twenty-four hours before or at the time of infection in their experiments prevented the development of paralysis.

In man more favorable results would be expected from the use of antitoxin than their work upon the guinea-pig indicates, for they were dealing with an early and malignant form of experimental postdiphtheric paralysis. This grave variety is, fortunately, rare in man. Further, they injected the entire charge of the poison directly into the tissues of the guinea-pig, while in man the toxin is doubtless elaborated more slowly. It may therefore be assumed that antitoxic serum, given at a somewhat later period than in their work upon guinea-pigs, would exert beneficial effects.

The fact that one unit of antitoxin prevents paralysis and saves life when administered timely, whereas 4000 units totally fails when delayed forty-eight hours, emphasizes the importance of using this sovereign remedy early.

THE TREATMENT OF DIABETES MELLITUS.

The *Practitioner* for July, 1907, contains an article on this topic by PARSONS. He concludes that:

1. There is no specific treatment for diabetes.

2. Drugs play at best only a subsidiary part in diabetic therapeutics.

3. Opium is the best antiglycosuric drug at present available. It may be administered in any stage of diabetes, but is most useful in severe cases, in which a rigid diet fails to remove all sugar from the urine. Dose, 1 to 5 grains of the extract.

4. Sodium salicylate and aspirin are valuable in mild cases of diabetes, and may temporarily completely remove glycosurin. Dose, 15 grains three times a day.

5. Jambul may be used as an alternative to sodium salicylate or aspirin.

6. A positive ferric chloride reaction indicates the daily administration of bicarbonate of soda in doses of 150 grains and upwards.

7. The intravenous injection of 35 ounces of a three- to four-per-cent solution of carbonate of soda affords the best chance of restoring consciousness in diabetic coma.

8. Constipation should be guarded against in all stages of diabetes.

SUDDEN CEREBRAL LESIONS: THEIR DIAGNOSIS AND TREATMENT.

GOWERS in the *British Medical Journal* of July 6, 1907, while not feeling very hopeful of therapeutic results in these states, thinks that in the immediate treatment some measures may be adopted in each lesion. A gouty state is common, and generally presumable, because age-imperfection of the organs generally leads to an approximate condition, and justifies some lithia or potash, combined with a diuretic, nitrous ether and spirits of juniper, to which some digitalis may be added —more if thrombosis is suspected than if hemorrhage is probable. The opposite rule should obtain with an aperient. If a clot has formed, only gentle action of the bowels should be secured; if a vessel has ruptured, an active purge is wise, such as croton oil, to lower the blood-pressure. Salines, it should be remembered, seldom alone act well in the horizontal posture; they need the aid of gravitation. Blood should be drawn to the extremities.

If there is reason to regard the lesion as thrombosis, little more can be wisely done. We have no practical agent to reduce the coagulability of the blood. Cit-

. ric acid is said to have this effect, and may be given as lemon juice in plenty of water. When hemorrhage may be confidently diagnosed, the treatment should be such as to increase the tendency of the blood to clot. Salts of calcium have this effect, and may be given in whatever form can be most promptly obtained. If the lactate or chloride is not at hand, common chalk may be converted to a soluble salt by any suitable acid that is available. Arsenic, by hypodermic injection, has a similar effect, and one-fifth grain of the arsenate of sodium may be given in a few drops of water. There is no reason to believe that small doses of iodide of potassium, which are constantly given at the early or later stage, exert the slightest influence.

But one measure is imperative in every case of either kind—physical tranquillity. It should surely be superfluous even to mention this, but experience shows, too often, that it still needs to be insisted on. Even in the slightest case, if the patient must be moved, it should be as little as possible, and he should be absolutely passive.

HEART DISEASE IN RELATION TO PREGNANCY AND LABOR.

In the course of an article on this subject in the *British Medical Journal* of May 25, 1907, BLACKER asks the question, What is the best treatment of these cases? Hicks and French maintain that the treatment of a case of pregnancy complicated by heart disease is to treat the heart disease, and that we need take no account of the pregnancy. And that conclusion being in large part a correct one, the author does not consider in detail the treatment of the heart disease, but discusses the obstetrical treatment, and mainly the question whether it is desirable to carry out any treatment of the pregnancy besides that directed to the condition of the heart.

If we have a patient suffering from heart disease complicating pregnancy, we will place the patient at rest in bed, treating the heart condition by such rest or by the administration of heart or general tonics. Above all things, we will insist upon absolute rest in bed in the worst cases. Is it, however, ever advisable or justifiable to treat these cases by the induction of abortion or by the induction of premature labor? Is the contention of Hicks and French wholly correct that we need treat the heart, and the heart only, or is it necessary sometimes to cut short the pregnancy? The author believes it is, and that in many cases the correct treatment is not only to treat the cardiac symptoms, but to relieve the patient of the strain produced on the heart by the pregnancy by the induction of abortion in some cases, or by the induction of premature labor in others. And he is of opinion that there are more cases in which the induction of abortion or of premature labor is good treatment than is generally supposed. It may be said that the immediate danger is increased by the induction of abortion or of premature labor, because the danger after delivery is greater than that during pregnancy and labor. Therefore we may from time to time, as in other severe diseases complicating pregnancy, such as albuminuria, meet with cases in which the result of inducing premature labor is markedly to increase the danger to the patient for the time being. But in the majority of cases, if such interference is really necessary, we will by its adoption improve the patient's condition, and not make it worse.

The following is a good illustration of the class of cases in which abortion should be induced: The patient, a woman aged thirty-two, had had two children and three miscarriages. She was suffering from double mitral disease, with marked dyspnea, edema of the lungs, cough, and hemoptysis, and was markedly anemic. Considerable improvement took place with complete rest in bed and medical treatment, but the cough continued to be very troublesome. The edema of the lungs did not clear up entirely, and after a time her general condition remained stationary. As she did not continue to improve, and as she was five months pregnant, so that she had

a prospect of another four months before her confinement could be expected, in which the strain on the heart would continue to increase, the author decided to induce abortion by the rapid method under anesthesia. The cervix was accordingly dilated under ether to No. 26 Hegar's dilator, and then to a diameter of three and one-fifth inches by Bossi's dilator, and the uterus emptied. A considerable degree of hydramnios was present. After delivery the symptoms rapidly improved, the cough ceased, the physical signs of congestion and edema of the lungs disappeared, and the general condition improved very markedly. In such a case as this the induction of abortion is justifiable and occasionally necessary. The author here recapitulates the reasons why in this case he induced abortion: first, because the patient had marked symptoms of failure of compensation as early as the fifth month, and secondly, that these symptoms, although they were improved by rest in bed and medicine, did not entirely disappear. He thought further that the strain of another four months of pregnancy, increasing, as inevitably it must, during the latter part of the term, would be too much for the weakened heart, and that therefore it was justifiable to terminate the pregnancy.

If we decide to induce abortion in one of these cases, then the best method is to give an anesthetic, to dilate the cervix, and to empty the uterus at one sitting. The class of case, then, in which abortion should be induced, is that in which ordinary medical treatment has produced no, or only very little, improvement in the general symptoms, and in which no further improvement in the cardiac condition is likely, so that it is essential that the strain of a long pregnancy should be avoided if possible.

The following case is an illustration of the class in which premature labor may be induced: The patient was twenty-five years of age, a primipara with double aortic disease. She had some edema of the legs, was markedly anemic, and had severe syncopal attacks from time to time. With treatment by rest in bed and medicine she improved considerably, but when she got out of bed and about she had further syncopal attacks, so that her condition was not quite satisfactory. He tided her over her difficulties as long as possible, but at the thirty-eighth week the author determined to induce labor because the syncopal attacks were rather frequent, and it was therefore thought best to put an end to the pregnancy. A Champetier de Ribes's bag was introduced under an anesthetic, and when the first stage was completed the patient was delivered under ether with forceps. The child is alive, the mother did well, and at the time his article was written had lost all her cardiac symptoms.

Some authors hold that the induction of premature labor increases the danger to the patient; but the writer does not think that this is so, because if we induce premature labor we can make arrangements to carry out the delivery in the best conditions and with the advantage of skilled assistance. He thinks, then, that in the case of patients who do not improve under ordinary medical treatment, it is wiser to terminate the pregnancy rather than to let the patient go on to full term. This does not agree with the conclusions of Hicks and French, based upon the obstetrical histories of their 300 cases. They contend that the induction of premature labor is not necessary, since the great majority of their patients bore a considerable number of children with little or no danger. No doubt this is true of the general run of cases, just as it is true that the majority of cases of valvular heart disease complicated by pregnancy do very well with ordinary medical treatment. But we will meet with exceptional cases from time to time which require not only medical treatment, but, in order to lessen the strain thrown upon the overburdened heart, require further than this the induction of abortion or of premature labor.

The treatment of the condition at the time of the patient's confinement is of some importance. We must guard against the risk of cardiac failure, and if there are

signs after the delivery of the child that the right side of the heart is overdilated, we should encourage postpartum hemorrhage, or we should actually bleed the patient. Such a mode of treatment undoubtedly yields good results where there are symptoms of overdistention of the right side of the heart and consequently danger of paralysis. Where the patient is pallid and the pulse very small, with no signs of overdistention on the right side, and where it is probable, therefore, that the blood is accumulating in the abdominal veins, the intra-abdominal pressure should be maintained at the time of delivery by the use of a sand-bag or a tight binder, and appropriate means must be taken to stimulate the heart's action.

There remains for consideration only one question, and that is the truth of the dictum laid down in many books that a patient with heart disease should not marry, or if she does marry she should not have children, or if she does have children she should not suckle them. Such a dictum, it will be agreed, is too sweeping. The majority of women with heart disease pass through their pregnancy and confinement with perfect safety and have no symptoms, and therefore it is not right to say that a young woman who has heart disease should never marry. If her heart disease is compensated, and there are no symptoms, she may marry. Cardiac failure probably will occur sooner or later, as Hicks and French point out, whether she becomes pregnant or not. The bad effect produced on the heart by pregnancy is, on the whole, not sufficiently marked to justify us in advising a patient strongly that she should not marry. It is true that if she marries it will be better for her not to have children, and it is true that if she does have a child she should not suckle, but it is not right that a woman with heart disease should be forbidden to marry. Although the danger is undoubted, and the mortality is as high as 12 per cent, yet it has been exaggerated, and the majority of these patients do perfectly well, and run no very great extra risk. So that a young girl with heart disease which is compensated, and which is not complicated by any accompanying disease of the lungs or other viscera, may be permitted to marry. Her heart will probably fail sooner or later, whether she marries or not, but whether the time of the onset of such failure of the heart will be precipitated by child-bearing is a matter which admits of argument. There probably will be some precipitation, but it is doubtful whether the risk of this is so marked as to justify us in denying her the right to marry.

ATOXYL.

LAFAY (*La Clinic*, May, 1907) calls attention to the great therapeutic value of atoxyl. It is not a new product, having been obtained in 1863 by heating an aniline arseniate, and contains 29 per cent of arsenic. It is largely used for cutaneous affections, for acne, lichen rubra, psoriasis; it gives excellent results in acne vulgaris, and cures psoriasis completely. In chronic eczema, alopecia, and urticaria the results have been negative. Siegel obtained good results with atoxyl in anemic and chlorotic women, and claims that benefit results from its use in cases of valvular disease and niyocarditis. In neurasthenia, hysteria, and chorea it is valuable, and Basedow reports surprising results in a case of epilepsy for which he employed it. Its use is particularly indicated in tuberculosis, and tuberculous glands and scrofulous affections generally. Moller got results which deserve to be better known when he gave it in intravenous injections in a case of pulmonary phthisis; and the author reports a series of cases of phthisis which have benefited so much from its use that he is inclined to consider it in some sort a specific for tuberculosis. It may be given by the mouth, subcutaneously, or intravenously. When given internally atoxyl is not well tolerated by the stomach. Rhoden overcomes this difficulty by giving it in the form of a pill with ichthyo-salicyl. In the treatment of tuberculosis two pills are given daily, after two days three pills, and

after a week the dose is increased by one pill daily until six are taken. For subcutaneous injections a 5- or 10-per-cent solution of atoxyl is used, and an injection given every five days, beginning with 0.2 cg. and increasing the dose up to 0.8 cg. When the maximum dose has been taken for some weeks it should be gradually reduced. For the intravenous injection a 20-per-cent solution is used in doses of 0.2 gr. every second day for three or four weeks.

Atoxyl is considered less toxic than other preparations of arsenic; it should be watched with some care at first, since the degree of toxicity is not absolutely decided. Fourneaux has thrown some doubt upon the widely curative powers claimed for atoxyl; it is, however, an active and useful drug, and merits trial.—*British Medical Journal,* July 13, 1907.

CONSTIPATION IN INFANCY AND CHILDHOOD.

The *Clinical Journal* of June 19, 1907, has in it an article by CAUTLEY in which he gives the following advice:

First of all relieve the retention of feces, and secondly try and cure the habit. Treatment must be steady and persistent. In the simplest cases, in breast-fed infants, all that is necessary is attention to the diet and habits of life of the mother. The breast feeding must be properly regulated. The quantity of milk can be increased by a liberal supply of fluid, tonics, etc. The percentage of fat in the mother's milk can be increased by giving a liberal diet with an excess of proteid, by a moderate allowance of alcohol, and by prolonging the intervals between each feeding. Liberal diet must be combined with plenty of exercise, otherwise the percentage of proteid in the milk is also increased. Sometimes the administration of a teaspoonful of cream in warm water twice a day, before feeding, is a sufficient remedy. For a bottle-fed infant a suitable milk mixture, containing a high proportion of fat and sugar, must be given. Water should be given freely between meals, and after a few months of age malted food or oatmeal may be added.

The habit of evacuation at the same hour daily must be taught to infants. At first it may be necessary to stimulate the action of the bowels by the introduction of the oiled tip of the finger, the nozzle of a syringe, or a soap suppository into the rectum. If these simple measures are insufficient, the following drugs may be used: manna, added once or twice a day to the bottle; sodium phosphate or citrate, gr. v. to x, or sulphur gr. j, added to each bottle, or fluid magnesia. If the stools are chalky and white, one or two minims of the tincture of podophyllin may be given two or three times a day. Peristalsis can be encouraged by abdominal massage along the colon, in a circle round the umbilicus, for five or ten minutes three times a day. The skin should be rubbed with oil to prevent excoriation. During the first year the palm of a warm hand should be used. After that age the finger-tips may be applied along the course of the large intestine, from the cecum to the sigmoid flexure, and no oil need be used. Soap suppositories are comparatively harmless, and consist of one grain of soap to five minims of oil of theobroma. They should be well oiled before insertion, and used night and morning. Small glycerin suppositories, gluten suppositories, or a simple cone of oiled paper are of similar value. If immediate relief is needed, from one-half to one drachm of glycerin in half an ounce of water should be injected into the rectum; it acts quickly and efficiently. Enemata of one-half to one ounce of cold water, soap and water, salt solution, or olive oil are useful. After the age of two years, medicated suppositories, consisting of 1/24 to 1/12 of a grain each of aloin, extract of belladonna, and extract of nux vomica, may be used night and morning. The continued use of suppositories or injections, whether of soap or glycerin, is not advisable, for it may set up catarrh of the rectum.

Internal medication is often necessary, but should only be used for chronic and severe cases. Manna, aloes, and sulphate

of soda may be given until two liquid stools are passed daily. This mixture should be given for two or three weeks and reduced slowly. At six months of age three to five minims of tincture of aloes, or the tincture of aloes and myrrh, may be given three or four times a day, combined with one minim of tincture of belladonna and fifteen minims of syrup of ginger. Tincture of nux vomica should be added if peristalsis is defective. To this mixture may be added ten grains of sodium sulphate or phosphate, or some syrup of senna. The syrup of senna, rhubarb, and figs are also useful. Drastic purges should be avoided. The usual dose of castor oil merely empties the bowel temporarily and is followed by recurrence of the constipation. For hard fecal masses an injection of warm olive oil, from one drachm to half an ounce, should be given, and followed in six hours by an enema of one to six ounces of soap and water, or salt solution. Older infants, say at two years, may have an injection of turpentine oil one drachm, olive oil two drachms, and the yolk of one egg, well mixed and added, with constant stirring, to four ounces of warm water. Injections must be given gently and slowly, and stopped at once if pain is produced. Mechanical aids, such as a bent hairpin, a scoop, or a teaspoon, must only be used by a medical man.

NOTES ON THE TREATMENT OF APOPLEXY.

PALMER states in the *Clinical Journal* of June 19, 1907, that there is a most important warning, which must be expressed in terms of negation: Never let an apoplectic patient lie on his back. This is, perhaps, the most important indication of all, and one that is not yet sufficiently recognized. The supine position is one which will, in most cases, bring a case of apoplexy to a rapidly fatal conclusion. It is in this position that stertor is most marked, and stertor is always an indication of danger, for apnea follows in its wake. We have already cerebral asthenia, and we can do without this additional complication. We cannot always relieve the asthenia, but we can in most cases prevent the apnea and keep the case uncomplicated, and this will make all the difference between the life and death of the patient. The accession of apnea will destroy the only chance of life remaining.

Dr. Robert Bowles, one of the original discoverers of the Marshall Hall method of resuscitation, has for the last fifty years been making observations on stertor in this connection; on animal dissections, on the dead body, on the healthy living body, as well as in pathological conditions—especially apoplexy and allied states—in cases of drowning and of artificial anesthesia. In season and out of season he has constantly insisted on the importance of this symptom in apoplexy, and the author thinks that any one who has read his work on the subject will be convinced of the correctness of his conclusions. He admits that when he first saw the account of these observations, about thirty-five years ago, he was not enthusiastic about them. He looked upon apoplexy as a fatal disease, and it did not appear to him to be a matter of any very great importance whether an unconscious patient lasted four days or only three. But he has seen recoveries since then, more or less complete, and is convinced that the exclusion of apnea and the encouragement of blood aeration by preventing stertor are important factors in the result. It would be impossible here to go through the various experiments and demonstrations; they are set forth in the work referred to. The general principle is obvious. Stertor means, of course, some obstruction to the entry of air into the lungs. In hemiplegia we get paralysis, on one side, of the muscles of the palate. When the patient is on his back the paralyzed muscles allow the tongue to fall back and diminish the opening into the larynx. Stertor ensues, aeration in the lungs is obstructed, the supply of pure arterial blood to the brain, now more than ever necessary for the restoration of its functions, is prevented, the backward pressure causes still further distention of the

cerebral veins and sinuses, cerebral auto-intoxication takes place, and the patient dies.

If the patient is now placed on the sound side, there is often little or no improvement. The paralyzed muscles fall down from the affected side and diminish the size of the opening as before, though perhaps to a less extent, and the stertor continues. The patient must therefore be placed on the paralyzed side; then the paralyzed muscles on this side will fall downward by gravitation, while on the sound side they will be held up by muscular action, and the largest amount of air possible will be able to enter the lungs. Stertor will cease; air will enter the lungs more freely; aerated blood will be conveyed to the cerebral arteries; while the cerebral veins and sinuses will be relieved of their distention.

In carrying out this treatment there is, of course, a very serious danger to be guarded against. If the patient lies long on the paralyzed side he will almost certainly have a bed-sore over the hip-joint. The position must therefore be changed at frequent intervals, by placing him for a time on the sound side; not on the back, if it can possibly be avoided. The tongue falling back toward the vertebral column causes far more serious obstruction than the palatine muscles, or the tongue even, falling to one side. In other words, pharyngeal stertor is more obstructive than palatine. Still, even a bed-sore, if it cannot be avoided, is a less evil than suffocation. In these people bed-sores form on the paralyzed skin with extraordinary rapidity. The author had it occur in one case, after which it healed up, and the patient recovered.

THE USE OF PILOCARPINE FOR THE RELIEF OF PRURITUS.

In the *Medical Record* of May 25, 1907, REID states that it does not seem to be generally recognized that pilocarpine has a specific effect in different forms of pruritus. Dr. Holstern used it in pruritus coincident with jaundice, and with happy effect. Since that time the writer has used it in pruritus vulvæ, pruritus associated with skin disease, and general pruritus without any assignable cause.

Pruritus seems to be a neurosis in some way allied to neuralgia, and has this in common with neuralgia, that it is periodic and may occur without any immediate cause. In pruritus vulvæ the writer has found the same benefit from pilocarpine in cases occurring with and without diabetes. The ordinary dose is one-quarter grain, to be given only when the itching manifests itself, and is not to be repeated unless the itching returns. It is well to begin with one-eighth grain, inasmuch as some patients are very susceptible to the drug.

It may be of interest to note a case of pruritus occurring in a medical student at the time of his final examinations. Without the use of pilocarpine he would have found it impossible either to think or write. In this case 1/120 grain of atropine in conjunction with the pilocarpine prevented the sweating. In all of the cases referred to pilocarpine was given by the mouth.

PRIMARY LIGATION OF THE VAS DEFERENS BEFORE REMOVING THE TESTICLE.

WALKER (*Boston Medical and Surgical Journal*, March 28, 1907) states that a recent study of genito-urinary tuberculosis has convinced him that during the excision of the tuberculous testicle the handling of this organ is likely to force tubercle bacilli into the abdominal portion of the vas or drive the organisms into the blood or lymph vessels and thus produce a general infection. In order to eliminate this danger an incision should first be made high in the groin and the spermatic cord ligated. After this the incision should be enlarged and the testicle brought up; the cord can then be cut below the primary ligature, and afterward such remaining part of the cord as desired cut off. The same procedure should be carried out in operating for malignant growths of the testicle.

SUCCESSFUL REDUCTION OF DISLOCATION OF THE CERVICAL VERTEBRA.

SHERMAN (*Boston Medical and Surgical Journal,* April 25, 1907) reports two cases of successful reduction of dislocation of the neck. The first case was that of a woman who had lifted a kettle of water from the stove with the left hand, at the same time turning her head quickly to the left. Her head remained in this position and was inclined somewhat to the right. Under chloroform anesthesia the head was tilted backward diagonally to the left and then rotated to the right. There was a distinct click as the reduction took place. On recovery from the anesthetic the patient was able to turn her head slowly in every direction. No apparatus was applied, but the patient was kept in bed with caution as to movements.

The second patient had a history somewhat like the first, as she was carrying a heavy weight when the accident occurred. The face was turned and tilted somewhat to the right. When relaxed under chloroform the head was tilted diagonally backward and to the right and then rotated to the left. No click was heard, but the head was then freely movable in all directions. The neck was painful and tender for a few weeks, but recovery was complete.

The author quotes Walton's description of the mechanism of dislocation of the neck and his suggestion as to reduction.

PUS TUBES IN THE MALE.

BELFIELD (*Medical Record,* May 4, 1907) states that the infections of the seminal tubes are three—the gonococcus, pyogenic bacteria, and the tubercle bacillus. As the last induces suppuration only with the aid of the pyogenic bacteria, it will be omitted from consideration as a cause of pus tubes in the male. The pus infections of the seminal tube are extensions from the deep urethra; they result from (1) gonorrhea, (2) stricture, (3) prostatic concretions, and other causes of prostatic suppuration in middle-aged and elderly men.

Invasion of the seminal vesicle by the gonococcus induces the symptom-complex usually considered indicative of prostatic abscess—frequent and painful urination; complete retention of urine, terminated by a sudden discharge of pus, often an ounce or two, into the urethra. These phenomena are usually due to abscess formation in the seminal vesicles, as emphasized by Lloyd fifteen years ago; or in the utricle, as shown by Belfield twelve years ago.

A typical gonorrheal abscess of the utricle was recently observed by Drs. L. E. Schmidt and Belfield; the symptoms were those usually ascribed to prostatic abscess, but the fluctuating swelling was greatest midway between the lateral borders. With the finger-nail the abscess was incised from the rectum; after the escape of perhaps an ounce of pus, the lateral lobes and median groove were distinctly felt.

Stricture of the bulbous urethra favors pus infection, which, beginning in the poorly nourished tissues, extends backward through the deep urethra into the seminal duct and vesicle, at times even to the epididymis. As the infection may extend forward in the urethra also, and thus produce a pus discharge from the meatus, the entire clinical picture simulates a gonorrhea, and by physicians who neglect to examine the pus with the microscope it is often so called—to the disgust and indignation of the innocent patient.

The non-gonorrheal infections of the seminal tube in men over forty years of age are, like everything else causing bladder symptoms in these patients, labeled "prostatic hypertrophy;" and they do indeed cause the same symptoms, including even complete retention of urine. These are the cases of alleged prostatic enlargement that were benefited by vasectomy, when that operation was popular. The prevalent rage for prostatectomy causes the unfortunate subject of vesiculitis to be accused of harboring an enlarged prostate and to be condemned to prostatectomy. A typical example of this common ailment of

elderly men was a patient seventy-two years old, seen with Dr. Henrotin nearly three years ago. He suffered from the usual symptoms of "cystitis" for several weeks, culminating in complete retention of urine. The left seminal vesicle was distended and tender, the prostate merely swollen; incision of the abscess was declined, but the pus traversed the vas, causing an abscess of the epididymis. When this was opened and the vesicle thereby drained, the bladder function was completely restored and the patient has had no urinary trouble since. A case practically the duplicate of this was seen with Drs. Billings and Phillips about the same time. By all the canons of the general surgeon, both these old men should have been subjected to prostatectomy, though their prostates were in no wise to blame, unless indeed because of the irritating presence of prostatic concretions, universally found in men past middle age.

The pus infections (non-gonorrheal) of the seminal tube, especially in elderly men, constitute an almost wholly neglected field; the obvious explanation is that already indicated, namely, the practical identity of symptoms with those caused by infections of the prostate and bladder, from which pus tubes have not been clinically distinguished. One whose attention has never been directed to this topic will be surprised to discover the number of cases treated for cystitis, prostatitis, and "prostatic enlargement" whose lesions are really pus infections of the seminal tube and vesicle. The differential diagnosis is by no means difficult; the usual mistake in diagnosis arises from the habitual failure to recognize pus tubes as the cause of pyuria, frequent and painful urination, and partial or complete retention of urine.

Gonorrheal infection of the seminal vesicle, on the other hand, has been frequently identified since Lloyd called attention to its occurrence and advised incision of the suppurating vesicle with a long-bladed knife through the perineum. About the same time Dittel, the father of perineal prostatectomy, used the incision which he had devised to attack the prostate for exposing and incising gonorrheal abscesses of the seminal vesicle. Dittel's operation was later modified and developed by Fuller, who has reported seventy-four cases in which he has attacked the vesicle for suppuration and other lesions through this extensive perineal dissection.

The Dittel-Fuller operation is admittedly severe and bloody, entailing risks and requiring weeks of confinement to hospital. It seems justified, if at all, only in cases of perivesiculitis, since the immediate relief sometimes demanded by pyovesiculitis is furnished far more simply and safely by incision with a cautery knife from the rectum. (The cautery is preferable to the knife for this incision, because it avoids bleeding, averts infection, and prevents premature closure of the wound.) But a much simpler and more effectual medication of the seminal tube and vesicle is secured by the operation that Belfield has devised and practiced, which can be done in the office. It consists essentially in opening the vas, stitching the cut edges to the skin, and injecting through a curved hypodermic needle any chosen solution into the proximal end; this liquid traverses the vas and ampulla and enters the seminal vesicle. By digital massage from the rectum the injected liquid can be expressed into the urethra if desired. Through the fistula daily injections of the vesicle can be made so long as seems necessary, after which the fistula is easily closed. Complete transverse section of the vas may safely be made, if preferred, since anastomosis of the cut ends and restoration of the lumen is easily accomplished. For injections through the vas the solutions commonly employed in the urethra are used; but the first injections should not exceed thirty to sixty minims, lest spermatic colic and retention of urine be provoked. Both of these effects the writer witnessed in his earlier work; the spasmodic contraction of the vesicle seems to cause spasm of the bladder sphincter, an item of both clinical and physiological interest. The influence of the seminal ducts

upon the bladder and its sphincter suggests an explanation of the cause and cure of "irritability" of the bladder in some cases.

CANCER—TREATMENT WITH PANCREATIC EXTRACT.

It has been shown that the glycogenic process is disturbed in cancerous individuals, and ODIER (*Revue Médicale de la Suisse Romande,* No. 3) finds that whereas the blood of healthy persons loses in specific gravity if placed in the oven for two hours, this loss being due to loss in glycogen content from hemic ferments, the blood of cancerous persons has always a high specific gravity from the large amount of glycogen always present, and there is no loss in specific gravity when it is kept at body temperature, showing absence of the usual glycolytic ferment.

He attempts to remedy this defect in the chemistry of the organism by giving injections of pancreatic extract. Backer has attempted to reach the same end by administration of yeast. Odier finds that the injections have a very marked influence on all recent growths, on metastases and recurrences, but very little effect on old or large tumors. The pain is always markedly influenced, and two cases of inoperable cancer of the breast were able to give up morphine after two injections. The only recent tumors not favorably influenced are those occurring in women long after the menopause, when the sexual organs are degenerated. There is some connection between the glycolytic function of the pancreas and the genital function.

HEMORRHOIDS—TREATMENT BY INJECTION.

The objections which have been raised to the injection of strong solutions of carbolic acid in hemorrhoids are answered by VULLIET (*Revue Médicale de la Suisse Romande,* No. 3), who claims that the method is without danger and that the objections apply only to other forms of vascular tumors, goitres, etc.

The method is only applicable to recent or inflamed growths, and not to old, indurated, quiescent masses. If the anus is fully dilated and the mass of vessels brought out into plain view, it is easy to introduce the needle so that the point reaches any part desired. The needle is introduced into each nucleus successively, and one or two drops of 80-per-cent carbolic acid slowly injected. The strong carbolic acid instantly coagulates the blood and tissues in the part, and thus prevents the absorption of the liquid, or its escape into the circulation. Any size mass may be treated, even those as large as an adult fist. The pain is slight.

FRACTURE OF THE LOWER END OF THE HUMERUS—OPERATION.

When the fracture is without displacement one may employ massage and passive motion from the first, the patient wearing simply a bandage for protection. Immobilization in extension is indispensable if there is lateral deviation, internal or external. These words of Rieffel are quoted by HUCHET (*Presse Médicale,* No. 24) at the beginning of his article.

Immobilization is to be condemned, as "motion is necessary to the life of the tissues." If the fracture cannot be reduced or cannot be held without splints, if the joint surface is involved, or if a nerve is injured, it is best to operate, and fasten the ends of the fractured parts. Otherwise a stiff joint is almost inevitable. The best method is to expose the elbow as required, and after reducing the displacement to bore a fine hole through the fragment and into the shaft of the bone. This drilled canal serves to guide a metal pin which is driven into the bone. Steel is to be preferred, and need not be removed unless a fistula forms. A single pin suffices to hold fractures of the condyles and epicondyles, while three are required for Y fractures. If the fracture is transverse and some distance above the joint it may be better to screw a silver plate on the bone. If there is trouble in the function of the ulnar nerve after such fractures, the nerve should be exposed and freed from adhe-

sions or pressure, etc. The most important point, however, is to deepen the groove behind the inner condyle, in which the nerve is protected from traumatism.

COMPRESSES LEFT IN THE ABDOMEN—PREVENTION.

A novel suggestion is made by WECHSBERG (*Centralblatt für Gynäkologie,* No. 12), who claims that no method of counting or other means of accounting for compresses is certain to guard against error as long as men are human.

He attaches each compress or piece of gauze, used after the abdomen is opened, to the wire basket in which they are sterilized. Only large compresses of several layers of gauze are used, and to each compress is attached a piece of narrow tape, over six feet long. The free ends of the tapes are passed through the meshes of the wire basket and tied together. The basket may be placed on the floor under the table. The tapes are not annoying after one is used to them.

TREATMENT OF HEMOPHILIA WITH ANTITOXIN.

A boy of eleven years, descended from a hemophilic family, forms the basis of a communication on this subject by BROCA (*Presse Médicale,* No. 24).

The boy had a severe hemorrhage, following the loss of a milk-tooth. The alveolus was tamponed with a piece of gauze saturated with antidiphtheric serum, and 20 cubic centimeters was administered subcutaneously. The bleeding ceased promptly and did not recur.

ABDOMINAL SECTION—GRIDIRON INCISION.

The praises of the gridiron incision are loudly sung by BARACZ (*Centralblatt für Chirurgie,* No. 12), who prefers the modification of Weir, for most cases.

This is an incision several inches long, running from and at right angles to a line drawn from the anterior superior spine of the ilium to the umbilicus. It may extend slightly above this line, and run parallel to the fibers of the external oblique. This muscle is separated with a dull instrument, and the fascia below cut in a line parallel to it. The incision is continued into the sheath of the rectus muscle, which is pushed aside. The internal oblique and transversalis are split, as was the external oblique, parallel to their fibers, and the peritoneum incised as necessary. This gives a much better oversight of the parts than the original McBurney incision, and is also useful for hernias, incarcerated inside the neck of the sac, for all operations on the cecum, and especially for artificial anus, as the crossed fibers form a sort of sphincter.

BREAST AMPUTATION—AFTER-TREATMENT.

To avoid the stiffness of the shoulder, which prevents the arm from being raised, after amputation of the breast, EWALD (*Centralblatt für Chirurgie,* No. 14) recommends the following dressing:

After dressing the wound as usual, but with the arm free, the patient is put to bed and the hand of the affected side is folded around a short stick of wood, and held closed by a bandage. The hand is then attached to a support which extends out from the head of the bed, so that the arm is about horizontal, and the forearm bent up. The elbow is supported by a plaster strip around the support. The position exercises traction on the fold of the axilla and thus prevents the collecting of blood in the wound. The arm is left still for three days, and is then lowered for a few minutes at a time until it can be moved without danger of opening the wound. When the patient rises the arm is fixed across the body while she is up, and suspended as soon as she returns to bed. The dressing has not been found especially uncomfortable, and the arm can be raised to the head at any time after the dressing is removed.

PLACENTA PREVIA—TREATMENT IN PRIVATE PRACTICE.

Immediate delivery after the first hemorrhage is recommended by FUETH (*Centralblatt für Gynäkologie,* No. 12), as there is great danger of secondary hemorrhage or septic infection with other methods of treatment, unless the patient can be watched continuously until delivery.

The liability to placenta previa bears no relation to the age of the mother nor to the number of previous conceptions. The first hemorrhage usually occurs some time before labor, and it is apt to recur if left to nature. Of the cases without immediate medical aid, one-third died. Of 726 cases only 23 had no hemorrhage before labor pains occurred. Treatment with tampons gives great danger of infection in private work, and also of repetition of the bleeding when no physician is at hand. It is therefore best to induce labor, preferably by introduction and inflation of a metreurynter, as soon as the diagnosis can be made. In hospital practice, where physicians are always at hand, it is perhaps safe to wait if the bleeding is not profuse or can be checked without difficulty.

CARCINOMA—UNEXPECTED RECOVERIES.

In several instances recovery has followed palliative operation performed for the temporary relief of inoperable cancers, and WEINDLER (*Centralblatt für Gynäkologie,* No. 22, 1907) reports three such cases which have occurred in his own practice. All were tumors of the cervix and uterus.

In the first case a vaginal total extirpation was contemplated, but abandoned on account of the extensive infiltration found at operation. Instead, the womb was deeply curetted, and the cavity burnt with the actual cautery. At long intervals the wound was cauterized with pure carbolic acid. The wound healed promptly, and there has been no return of the growth for five years. The other patients exhibited similar lesions, but the firm adhesions of the uterus showed operation to be hopeless. Both were treated by removing as much of the tumor as possible with the sharp curette, and burning the wound deeply with the actual cautery. This was followed by the carbolic acid treatment. These cases have remained free from recurrence for four years. Czerny has cured a case by a similar procedure, using tampons soaked in 30-per-cent zinc chloride solution instead of the carbolic acid. The recovery of these cases is not yet satisfactorily accounted for. Various explanations are offered by different pathologists, but the effect of the cauterization on the blood supply seems to be the most important factor.

THE TECHNIQUE OF PANCREATECTOMY.

DESJARDIN (*Revue de Chirurgie,* No. 5, 1907) notes that heretofore affections of the pancreas have been treated by indirect operation rather than by measures dealing immediately with the diseased organ, and that the results are palliative rather than curative. The author notes that only twice have surgeons attempted complete pancreatectomy—Billroth in 1894 and Franke in 1901, and both had an operative success. Billroth lost sight of his case; the one operated on by Franke died five months afterward of generalized cancer, but without presenting any symptoms that he perished because his pancreas was taken out. There are many reports of partial pancreatectomies, but usually these were incidental to other operations. Partial resections of the tail of the pancreas have given seven cures and three deaths; partial resection of the head, seven cures and nine deaths; total resections of the head, two deaths.

Desjardin notes that a complete pancreatectomy necessitates the removal of the duodenum because of the intimate adhesion between these two organs and the common vascular supply. He observes that in some cases the upper part of the ascending colon is opposed directly to the right border of the ascending portion of the duodenum without interposition of the serous

membrane. It must also be borne in mind that the mesenteric artery and vein as they emerge from the lower border of the pancreas pass in front of the duodenum, and finally the middle colic artery must be respected which lies immediately behind the pancreas, which may readily be wounded. This accident has happened in the hands of the best operators. Moreover, the portal vein and the vena cava are placed so closely posterior as to bring them within danger of trauma. To avoid the ascending colon Berg makes, two finger-breadths to the right of the second portion of the duodenum, an incision three or four centimeters long running down to the origin of the transverse mesocolon. The peritoneal flap with its base attached to the duodenum is stripped up by the finger, thus exposing the right border of this portion of the gut. If the duodenum be stripped well forward the portal vein and the vena cava are readily avoided. The stripping should be accomplished with the finger and is usually easy, since there is a natural plane of gravity. This stripping is regarded by Desjardin as the key of the operation, since it enables the surgeon to explore the lesion to bring the organ to the surface and to avoid wounding the important vital structures surrounding it. This blunt dissection must be practiced gently in the third portion of the duodenum lest the mesenteric artery and vein which pass in front of this portion of the gut should be torn. It is noted that the operation can be done in two stages, the gastro-jejunostomy preceding the pancreatectomy. If the operation is done at one sitting it is held that for the saving of time an anastomosis button should be employed.

As for the technique there is placed beneath the lumbar region a roll pillow as in biliary operations; a long incision is made from the costal border of the middle line downward to just above the umbilicus, and obliquely downward from this point to the left and below the umbilicus. The belly having been opened the omentum is pushed to the left, and an incision is made through the peritoneum dorsally 2 centimeters to the right of the descending portion of the duodenum and parallel with it. The peritoneum is stripped up toward the central line as far as possible until the portal vein and the vena cava have been passed. The pyloric gastroduodenal arteries are ligated, the right border of the omentum ligated and divided, and the pylorus is sectioned between two clamps. The duodenum thus freed is stripped downward, exposing the common duct, which is also divided between two clamps as low as possible if it is dilated, near the cystic duct if it is not dilated. As soon as the common duct is divided the duodenum is thoroughly freed except for the insertion of the mesocolon, which should be cautiously stripped a little for the purpose of disengaging that portion of the gut lying just beneath the mesocolon. In this blunt dissection the superior mesenteric vessels are encountered and the denuded jejunal angle is reached; the clamp is placed on the bowel at this point and it is divided. The duodenum is now held by the pancreas. On the body of this organ a clamp is placed obliquely, directed upward and to the right toward the pylorus in order to avoid injury of the colic artery. The gland is now sectioned, leaving in the wound a clamp on the pylorus, another on the common duct, another on the duodenum, and a fourth on the pancreas. All bleeding points are secured, and the stomach is closed by a double suture, after having placed through the pyloric opening and in proper position a Murphy button for an anastomosis with the jejunum. The sectioned end of this gut is then drawn up to the posterior wall of the stomach in order to make a terminal anastomosis, but on accomplishing this procedure two Boari buttons are placed in this portion of the gut. The jejunum is then implanted into the stomach by securing a Murphy button in its end and pressing the latter into the button already secured in the stomach wall. The common duct is then anastomosed with the jejunum by means of a Boari button, all bleeding points of the sectioned surface of the pancreas are sutured, the canal of Wirsung is freed, and it also is

anastomosed into the jejunum by means of a Boari button. This anastomosis is, of course, impossible unless the duct of Wirsung is dilated—indeed, the section may be carried so far back that two or three very minute ducts are all that can be found. Under such circumstances the cut section of the pancreas is inserted into the jejunum. This has experimentally been followed by disastrous results, but probably because the cut section projected into a portion of the intestine through which was passing the ordinary intestinal contents.

Desjardin proposes the following method for avoiding ascending infections: About 30 centimeters of the jejunum is drawn out and the bowel then divided between two clamps. The mesentery is then freed to such an extent as to permit without torsion complete mobility of the loop thus excluded. Into this mobilized loop is implanted the sectioned end of the pancreas, or at least that portion just above its ductus, and an anastomosis is made between it and the gall-bladder. One end of this loop is then implanted into the jejunum just below the insertion of the latter into the stomach. This excluded loop thus receiving the bile and pancreatic fluid is then joined to the jejunum just below its implantation into the stomach by lateral anastomosis.

CHLOROFORM SHOCK—LESSENING BY TRANSFUSION.

A long series of experiments made by BURKHARDT (*Archiv für klinische Chirurgie,* lxxxii, 4) led to the interesting discovery that although an infusion of salt solution given intraperitoneally, after chloroform narcosis, had no beneficial influence, a similar infusion given before narcosis lessened the time of recovery, shortened the period of albuminuria, and made over twice as much chloroform necessary to produce the death of the animal.

The experiments were performed on dogs, 250 to 300 cubic centimeters (8 to 10 ounces) being given intraperitoneally ten to twenty minutes before or five to ten minutes after the chloroform. Although the animals injected after the anesthesia excreted a very much greater amount of urine than those not treated, the excretion of the chloroform was not influenced. The dogs to which the injection was given before anesthesia passed a slightly increased amount of urine, but this contained much more chloroform. The excretion of the chloroform in the urine ceased several hours sooner than in the controls.

These results are explained by the fact that the principal element in the shock of chloroform is fall of the blood-pressure, and this is prevented by the use of salt solution before but not by its use after the anesthetic. In cats a mixture of air with 1 1/5 per cent chloroform causes a steady and progressive fall in the blood-pressure, which ends in the death of the animal. If a solution of salt is given immediately before narcosis, this concentration is insufficient to produce a marked fall. If an infusion is given in the course of a narcosis, it is followed by an immediate and marked rise in blood-pressure. For the last four months all patients operated on in chloroform narcosis have been given a hypodermoclysis of 1500 to 2000 cubic centimeters (1½ to 2 quarts) fifteen to thirty minutes before the operation. The fall of blood-pressure was very much lessened, the pulse good, the recovery from the anesthetic prompt, and after-phenomena—headache, vomiting, weakness, etc.—absent. There was no tendency to an increased amount of bleeding.

PRIMARY TYPHLITIS WITHOUT APPENDICITIS.

MCWILLIAMS (*Annals of Surgery,* June, 1907) in a consideration of this subject reaches the following conclusions:

Primary, acute, or chronic typhlitis occurs independently of appendicitis, dysentery, tuberculosis, actinomycosis, or cancer, and is idiopathic in origin or depends on coprostasis.

Primary typhlitis, the appendix being normal, may lead to perforation, with the

formation of perityphlitic abscess or general peritonitis.

The symptoms of primary typhlitis are usually identical with those of appendicitis, and the indications for operation are similar in the two conditions.

Primary typhlitis is rare in comparison with the frequency of appendicitis.

The differential diagnosis of inflammatory typhlitis from tuberculosis, cancer, and actinomycosis may be impossible at operation, in which case a section of the cecum should be removed for microscopical examination.

The recurrence of symptoms after removal of the appendix may be due to attacks of typhlitis, the treatment for which consists in the regulation of the diet and the use of oil enemata, etc.

The danger of a primary typhlitis consists in the liability to rupture of an ulcer and to the development of appendicitis.

Purgatives are contraindicated in inflammations of the right iliac fossa because of the danger of rupturing an ulcer in the cecum or appendix, or of breaking up the adhesions about an abscess.

The treatment of primary typhlitis consists in laparotomy, removal of the appendix, closure of any perforation if possible, invagination of any threatening perforation of the cecum, drainage of the right iliac fossa, and the removal of a piece of the cecal wall for microscopical examination, if that be necessary to establish the diagnosis.

AVULSION OF THE TUBEROSITY OF THE TIBIA.

If the foot is suddenly arrested during a powerful contraction of the quadriceps, any of three accidents may result. The most common of these is transverse fracture of the patella, the next is rupture of the tendon, and LINKENHELD (*Deutsche Zeitschrift für Chirurgie*, lxxxvii, 1) states that avulsion of the tuberosity of the tibia is extremely exceptional. When it does occur it is almost always unilateral, the case reported in this article being the only bilateral one, as far as could be determined. It occurs almost always in persons from fourteen to twenty years old, frequently during gymnastic exercises. The case reported occurred in a gymnast of sixteen years, whose heel caught as he made a difficult jump from a spring-board. The accident resulted from a violent effort to save himself. The symptoms were those usually seen—immediate inability to stand or to extend the leg and extreme pain below the kneecap, followed shortly by swelling of the soft parts and effusion of blood. There was no effusion into the knee, which is often noted. Examination showed on one side the fragment torn free and drawn upward, and on the other side only partly free and turned forward like a hinge from its upper edge. *X*-rays confirmed the diagnosis, which is usually easily made. Treatment was operative, and recovery complete and prompt.

Of 29 similar cases reported, 23 were treated without operation; of these 11 had a perfect result, 7 were permanently more or less lame; in 5 the result is not mentioned. The duration of treatment was from two to five months. In all 6 of the cases operated on the functional result was perfect and the treatment lasted only from three to eight weeks.

The operation consists in exposure of the injured point, cleansing of the wound, and reposition of the fragment. If it is only partly torn away it may be held in place during healing by sutures through the periosteum. If these are not sufficient, a nail is driven through the fragment into the bone. The choice of incision and technique lies with the surgeon.

The liability to the accident is dependent on the imperfect union between the tuberosity and the shaft of the bone during early life. It is not certain whether the fragment develops from its own nucleus, although this is probable, or whether it grows down like a protruded tongue from the articular cartilage, but in any case the mechanism of the fracture is much the same as that of separation of an epiphysis. Complete bony union of the tuberosity occurs normally between the twelfth and twenty-fifth years.

THE PROPER SHOES IN AID OF THE TREATMENT OF FLATFOOT OR WEAKENED FEET.

BERRY (*American Journal of the Medical Sciences,* May, 1907) notes that the weakened foot goes through a series of changes, beginning with a disability known as pronated foot and ending with the deformity of rigid flatfoot, and that the term flatfoot cannot be used to designate all the forms of this trouble, but should be restricted to one phase of the progressive weakened condition.

A brace he considers necessary at times and nearly always beneficial. In order that it should have its full benefit it should be used in conjunction with a proper shoe. Strength comes with a brace or shoe only as it is properly used. Physicians can supply the apparatus, but the patients must cure themselves, since when the foot is put in proper position its ultimate strength and self-dependence rests absolutely upon active exercise, gymnastics, etc., upon the part of the patient.

Berry observes that a shoe should have a low heel with little or no spring to the sole, thus allowing the toes to come to the ground. It should be broad enough across the ball of the foot to give full play to the weight-bearing portion. The shank should be to the outer side of the shoe (where the weight of the foot comes) instead of in the middle, as in the ordinary shoe. The front part of the shoe should have the same angle of adduction as is normal to that individual foot. Care should be taken, especially in weakened feet, to keep the heels of the shoes built up and not to become worn off on one side.

Berry's method of getting a proper shoe made is as follows: He obtains an imprint of the foot by thinly spreading on a sheet of plate glass, by means of an ink roller, one part of lampblack, one part of powdered acacia, and three parts of water. The patient steps on this with his whole weight, thus blackening his weight-bearing sole. He is then instructed to raise his foot, and after the glass has been replaced by a sheet of rough white paper, to bring the foot down upon the paper and rest his full weight upon it. A pencil tracing is then made around the foot. When the foot is placed upon the paper care should be taken that it is held parallel to the other foot, since in that position the feet have a normal amount of adduction. An inner sole patterned after the last is now marked out with the imprint and the tracing as a guide. The shoemaker takes measurements as follows: (1) Circumference of the foot over the big and the little toes; (2) the ball of the foot; (3) the waist; (4) the instep; (5) the heel and the top of the instep; and (6) the heel. The measurements are also taken of the ankle and the top of the shoe. These measurements and the inner sole patterns are sent to the last-maker, who has a model last in stock from which he makes for the patient a similar last, but having an inner sole pattern and measurements corresponding to the patient's foot. The last is returned to the shoemaker for any little changes necessary to make a better fitting shoe. Separate inner sole patterns, measurements, and lasts have to be made for both feet of the patients.

There is usually some difficulty about the patient becoming used to the new shape, but in a few days the discomfort wears away and the freedom of motion and comfort consequent upon the use of properly constructed shoes soon cause him to become an earnest advocate of their use.

THE SURGICAL TREATMENT OF SPLANCHNOPTOSIS.

DAVIS (*Annals of Surgery,* May, 1907) notes that tenderness over all parts of the abdomen is one of the almost invariable symptoms of splanchnoptosis associated with a greater or less degree of autotoxemia and many nervous manifestations. Two anatomical changes are present: the weakened muscular wall of the abdomen, the stretched and attenuated ligaments. To these must be added dilatation, to a greater or less degree, of the stomach and intestines, and increased weight of the solid

organs, liver, kidneys, and spleen, due to the long-continued passive congestion. The tendency is for the trouble to become progressively worse, for the abdominal muscles to become weaker and thinner, the suspensory ligaments longer and more attenuated, the dilatation and enlargement of the viscera more pronounced.

The author does not believe that any case of extreme splanchnoptosis is ever cured by medical or mechanical treatment. Davis advises correction of gastroptosis by suturing the small omentum near its attachment to the smaller stomach curvature to the abdominal wall, as high as possible. He believes this operation quite as good as the one of shortening the lesser omentum, and it is very much easier to do and seems less likely to interfere with the circulation. Moreover, in many of these cases a hepatopexy is needed at the same time, and it is as well not to have the extra weight of the stomach and colon pulling down on the liver.

Coffey's method of suspending the stomach in a hammock by fastening the omentum to the abdominal wall seems rational and especially applicable when the lesser omentum is so weak as not to be equal to the task of supporting the stomach. It is possible this method may develop a kinking at the pylorus.

Gastroplication, in cases of dilatation, seems of little value. If the dilatation is dependent on descensus, when the stomach is raised to its normal position it will very soon regain its natural size.

If, on the contrary, dilatation be due to obstruction, it will recur unless the obstruction be removed.

Gastroenterostomy has been strongly advocated and gives fairly satisfactory results, though it does not cure the condition. After any suspending operation, several weeks in bed with vigorous massage of the abdominal wall and forced feeding after the operation are indicated.

In prolapse of the liver, Davis states that he is coming to depend more and more upon shortening the suspensory and round ligaments.

For prolapse of the transverse colon, Coffey's method for suspending the stomach in a hammock by suturing the great omentum to the abdominal wall is advocated, since incidentally it holds up the transverse colon.

Davis states that most contributors to surgical literature of splanchnoptosis ignore the existence of enteroptosis, or consider that suspending the stomach corrects the entire trouble without any effort directed to the small intestines. Usually this is probably true, but sometimes the mesentery is stretched to two or three times its natural length.

Davis in these cases shortens the mesentery very much as Beyea shortens the gastrohepatic omentum, except that, instead of folding the membrane on itself by tier suture, only one suture is introduced between the arteriæ intestinæ tenuis, and when it is tied it produces a reef. Many of these sutures are used, and as they are tied the mesentery is shortened as much as desired.

The putting in of these reefs is not nearly so formidable as it seems. A number of cases are reported, one of which will sufficiently illustrate the technique.

The stomach was drawn up into its normal position, and the lesser omentum near its reflection upon the stomach at its lesser curvature was fastened to the peritoneum (in a transverse direction) at the level of the ensiform cartilage by means of fine silk sutures. The stomach was not especially dilated, and gastroplication was not performed. The transverse colon was fully six inches from the greater curvature, the gastrocolic omentum having been greatly stretched. A tuck was taken in the gastrocolic omentum, being careful to avoid the vessels, and not allowing the sutures to penetrate more deeply than through the anterior peritoneal layer of the omentum. This shortened the distance between the transverse colon and greater curvature of the stomach to two or three inches. The small intestines were now brought forward and the mesentery found to be so much elongated that the loops of intestine could be raised four or five inches

above the level of the abdominal wall without undue tension. Beginning near the upper end of the jejunum, a loop was brought forward. To shorten the mesentery without interference with the intestinal blood supply was the problem before the operator. The isosceles triangles, bounded at the base by the attached border of the intestine and having for their sides the arteriæ intestinæ tenuis, branches of the superior mesenteric, were elongated, the distance from their apices to their bases being three to four inches. Anything might be done to shorten these triangles if there were no interference with the circulation at their borders. A long, slender needle, carrying No. 4 silk, was inserted near the apex of a triangle, penetrating the mesentery in one direction and brought through in the opposite direction at the center of the base near the attached border. The suture was drawn through and tied, forming a reef in the mesentery at this point and shortening it from two to three inches. After several sutures had thus been introduced and tied in contiguous triangles, close examination showed that the circulation was unimpeded. Sutures were thus used the entire length of the small intestine, not in every interarterial space, but almost that closely. Between the upper jejunum and the ileocecal valve ninety-two sutures were employed. There was no shock. The patient did well with the exception of some pain in the region of the loosened adhesions. There was no distention. The bowels moved the third day. The patient was up the twentieth day, and left the hospital August 9, exactly four weeks from the day of operation.

UNDESCENDED TESTICLE.

Barr (*American Journal of Surgery*, January, 1907) holds that undescended testicle is at all times a surgical condition which should invariably be operated on before puberty, preferably between the ages of ten and twelve years. Even after puberty operation is still indicated, unless the testicle is within the abdominal cavity, and there is entire absence of hernia. He notes that the spontaneous descent of the undescended testicle is uncommon after the first year of life, and that in double cryptorchis, unless the condition be relieved, sterility is certain. It is often associated with impaired virility. Moreover, he calls attention to the fact that undescended testicle is liable to accident and disease, and is frequently complicated by hernia which cannot be supported by a truss. He believes that by operation before puberty there is more than a reasonable hope that the testis will develop along normal lines. If it cannot be brought down into the scrotum it should be replaced extraperitoneally within the abdomen.

Barr indorses Bevan's method of complete separation of the processus vaginalis from the cord, and its division, together with separation of the posterior and parietal peritoneum from the spermatic vessels as they pass upward from the internal ring, with a further tearing across of all fascial bands in the cords.

LOOSE KIDNEY—CHOICE OF PROCEDURE.

Morris (*American Journal of Surgery*, January, 1907) aptly remarks that many of the loose kidney patients of earlier days have to look upon their operations in an altruistic way, and have to be grateful only for the opportunity which they had for furnishing negative testimony for the benefit of the profession and incidentally for the benefit of the public. He gives a most rational classification of the cases in which operation is not likely to prove serviceable, including in this patients in whom loose kidneys cause really no disturbance, those in whom the abnormality is associated with minor psychoses not dependent upon loose kidney, and those in whom a loose kidney is only a part of a panoptosis of the abdominal viscera.

Morris advises the trial of abdominal supporters in all cases in which there is any doubt about whether the low kidney is "Hamlet in the play of symptoms." This

failing he suspends the kidney by a kangaroo suture entering the lower pole of the organ, carried along the dorsum for half its length, then across the other side, then down and out near the point of entrance. The two loose ends are made fast to any convenient muscular attachment, thus hanging the kidney where it is wanted. The lower pole of the kidney is then packed with iodoform gauze in such a way that the retroperitoneal pouch is distended. Gauze in a narrow strip is employed, one end of which is left outside of the wound. The sheath of the quadratus lumborum muscle alone is sutured, and that allows good approximation of deeper structures. The fatty capsule of the kidney is trimmed, and the fibrous capsule is split along the dorsum in order to give relief from the inflammatory tension. At the end of a week the iodoformized gauze is removed, and no further attention is required, excepting change of outer dressings for neatness. The patients are gotten out of bed at the end of fourteen days.

In this operation the kidney is properly suspended, and the gauze, which at first acts as a drain, will later by the adhesions which it causes obliterate the retroperitoneal pouch and fix the nephrocolic ligament.

Morris states that the only instruments he uses for the operation are a pair of scissors and a needle. There are no vessels which require ligation. The entire operation can be finished in fifteen minutes, and he has done the double operation in that time without working hurriedly.

REVIEWS.

HYGIENE OF NERVES AND MIND IN HEALTH AND DISEASE. By August Forel, M.D. Authorized Translation from the Second German Edition by Herbert Austin Aikins, Ph.D. G. P. Putnam's Sons, New York and London. The Knickerbocker Press, 1907.

The subject-matter of the work before us is divided into three parts: the first of which deals with the Mind, Brain, and Nerves in their Normal Condition, the second with the Pathology of the Nervous Life, and the third with the Hygiene of the Mental Life and of the Nervous System. The chapters of the first part embrace Psychology, the Anatomy of the Nervous System, the Relation of the Mind to the Brain, the Physiology of the Nervous System, Embryology and Race History of the Nervous System, and are written in a popular though very lucid vein. On the whole these chapters are very acceptable. Bearing in mind the extremely popular character of the exposition, close criticism is perhaps not called for; generality of statement is to some extent indispensable in a popular presentation of so complex a subject. The chapters on the Pathology of the Nervous System are likewise extremely condensed and thoroughly popular in their exposition. They are notwithstanding very clear, and the definition of the subject must be exceedingly valuable to lay readers. As an instance of the character and lucidity of statement the following may be cited: "The general characteristic of the mentally diseased, as contrasted with those who are at least partially sound, is the lack of insight into the abnormality of their own condition. This rests on the alteration of the whole personality, and this in turn upon diffused changes in the functioning of the brain, as a result of which the outer world and other people impress it differently, and the patient attributes this change to them and not to himself."

A brief synopsis of the various nervous and mental diseases is given, the exposition being on the whole satisfactory. However, like many of the continental writers, the author is not in touch with the western conception of the great functional neuroses. For instance, he fails entirely to recognize neurasthenia as a fatigue neurosis and hopelessly confuses it with hypochondria. He declares, for example, that "hypochondria is the principal ingredient in the *omnium*

gatherum of diseases thrown together nowadays under the name of neurasthenia." His interpretation of hysteria is likewise to say the least peculiar. He recognizes the element of dissociation in hysteria, but evidences a curious conception in regard to the rôle played by the disease. Thus we are told: "As a result of such dissociations it is possible for some overpowering feeling or idea to call forth various kinds of permanent paralyses, cramps, anesthesias, hyperesthesias, pains and all sorts of other symptoms of disease, fits of rage, sexual abnormalities, inhibitions, or strong irritations; but also, on the other side, ingenious pieces of work, the healing of these very diseases, enthusiasm for the good, self-sacrifice, heroic deeds, and, in short, everything that the human brain can prevent or produce. Hysteria as a disposition of the brain is to some extent a two-edged sword. It engenders an untold amount of evil and many misunderstandings, and yet delivers from many sufferings—and is misunderstood by very many physicians. Hysterical persons, misled or otherwise badly molded, can become devils; but if they are well-fed or of noble nature, they are often angels or heroes, like the Maid of Orleans." The suspicion is here justified that the author does not clearly distinguish between hysteria and paranoid states.

The etiology of mental disturbances is briefly and satisfactorily considered. We are, of course, impressed by the overwhelming rôle which the writer attributes to alcohol, a position which his well-known views on the subject would lead us to anticipate.

The section on the hygiene of the nervous system is, plainly speaking, disappointing. The writer again expends much energy on the rôle played by alcohol, which we read patiently, hoping to be rewarded in subsequent pages by a scientific discussion of the important subject-matter of the chapter. However, we are met with commonplaces and platitudinous statements regarding habit, training, exercise, feeding, and sleep. In vain do we search for a consideration of the problems of nutrition and diet, of tissue metabolism and elimination, which bear so vitally upon nervous health. Instead we find the writer making a vigorous onslaught on medicines, electricity, hydrotherapy, and physical remedies in general, as though the function of a chapter on hygiene was the discussion of medical methods. Upon every occasion also we find the author extolling the value of hypnotism—again not a surprising thing in Forel, but none the less disheartening. It is strange that hypnotism, now almost universally admitted to be merely hysteria artificially induced and proved by over a century of trial to be not only useless but dangerous, should still find its advocates, and in this instance in the person of an alienist to whom practical experience with the insane must have proved its utter futility. We are further disagreeably impressed by the writer's ungenerous and unjustifiable attitude to physicians in general, and somewhat amused by the specious advice he gives to patients in regard to the selection of a "reliable mental and nervous specialist." "Moreover," he says, "one should set aside his fear of the alienist. The director of a state asylum with his fixed salary, and exposed as he is in his thorny place to enmity and calumny through the gossip and lies of uncured or only half-cured patients, is the very person to offer good security, for the ground on which he lives is not adapted to the growth of fraud. His knowledge of the abnormalities of the human mind gives him practical wisdom which is usually lacking with other physicians. Many so-called nerve doctors who are accustomed only to sanatoria, and have confined their studies to the spinal cord and peripheral nerves, suffer from the great mistake that they do not know the center of their own field, the brain and mental disturbances." A passage such as this needs no comment; on the face of the American reader we observe a quiet smile.

The book, as might have been predicted, is in many respects an admirable exposition of the elementary science of the subject, marred by a rabid antialcoholism, a vaunting of hypnotism, and a denunciation of other methods. F. X. D.

CORRESPONDENCE.

LONDON LETTER.

BY G. F. STILL, M.A., M.D.

The "silly season" is fast drawing to its close. Newspaper columns are still laboring to make bricks without straw. "Constant Reader" and "Indignant Citizen" are still pouring forth letters on "Slack Mothers" or "Are We a Stupid Nation?" But there are signs that London is coming home from its holidays. Editors are giving notice that "this correspondence must now cease," dusty blinds in West-end houses are beginning to be pulled up, and the loitering unemployed are watching for luggage-laden cabs which may bring them an honest penny for help in unloading. At the hospitals also, where whitewash and confusion have reigned supreme for weeks past, and beds have jostled each other in a disorderly crowd shrinking from the invasion of profane painters and polishers, order is now beginning to reappear, and medicine once more scorns to mingle with surgery, and each is reinstated in its separate ward.

With the first day of October students will return to the hospitals, and each medical school begins its session. This event is ushered in by an address delivered by some distinguished person, with a subsequent restorative in the shape of the "hospital dinner," a function which is patronized by the former students of the hospital, who turn up in force from all parts of the country. At St. George's Hospital, Dr. William Ewart will deliver the inaugural discourse, taking for his subject "Res medica, res publica"; at St. Mary's Hospital, Professor Osler is to be the speaker; at King's College Hospital, Dr. Allchin, the senior physician of Westminster Hospital, will speak on the present position of medical education in London. It is curious to note the change that has come over the spirit of the inaugural address: in years not very remote it was the custom for some member of the staff of each hospital to deliver the address, which was usually in part a formal welcome to new students, in part a homily to all the students, who were therein exhorted to follow all that is noblest and highest in the profession of their choice, and in general to walk worthily of their high calling. No doubt the interchange of speakers, so that each hospital has some distinguished visitor for the occasion from another medical school, has the advantage of promoting good fellowship between the hospitals, and it is good that these addresses should be made the occasion for public pronouncements on important questions concerning medical education or medicine in relation to the State. But still one cannot but feel that the old plan was good, and that there is only too little opportunity for teaching, from those whose years give them the claim to teach, the ethics and morals of medicine, which those who are just entering the profession can hardly be expected to learn by instinct; though indeed it would seem as if this were expected, for it is clear from the correspondence which only too frequently appears in our medical journals that on these points each medical man is a law unto himself, and his law is sometimes not altogether in harmony with that of his neighbor practitioner.

Considerable ill feeling has been aroused amongst the medical profession by the action of Parliament recently in passing the Notification of Births Bill. This measure, passed under the guidance of Mr. John Burns, the president of the Local Government Board, enacts that every birth shall be notified, and that any medical man attending the confinement or called to attend within six hours after the birth shall furnish a written certificate of birth to the local authorities within thirty-six hours after the confinement. The generous means by which this certification is to be insured is not the payment of a fee to the hard-worked doctor, but the infliction of pains and penalties if he fails to give the required document.

A remarkable case of gin-drinking at the age of three years and nine months

ended fatally in London this month. The child apparently drank nearly three ounces of neat gin, then vomited and became unconscious, with violent convulsions which lasted till death, about twenty-four hours after taking the gin. The parents declared that the child had never to their knowledge tasted alcohol before, but the medical evidence showed that there was cirrhosis of the liver, which was stated to be such as is produced by chronic alcoholism.

It is not often that a member of our profession lives to see his hundredth year, but the venerable figure of the Emeritus Registrar of the Royal College of Physicians of London is still to be seen walking about at his suburban home in his hundredth year. Sir Henry Alfred Pitman was born in 1808, and took his Bachelor of Arts degree at Cambridge in 1831; he was physician to St. George's Hospital from 1857 to 1866, and was knighted by Queen Victoria in 1883.

A medical man named Dr. Hirst met with a tragic end at Leeds a few weeks ago. He had been attending a man for mental disturbance, and after some delay it had been decided to certify the patient as insane and place him in an asylum. Apparently in the course of examining his patient the doctor had tested the knee-jerks, which the patient described as blows upon the kneecaps, causing "terrible epileptic fits." He attributed various sinister designs to the doctor, said that the doctor had killed him and deserved punishment, and, it is thought, may have gathered that the doctor had decided to put him under restraint. However this may be, one morning at six o'clock he knocked at the doctor's door, and when the doctor himself opened the door to him the lunatic fired two shots from a revolver, killing the doctor instantly, and then turned the revolver on himself and ended his own life. The doctor was twenty-eight years old and had been married only three months.

The mention of longevity in our profession reminds me that at the opening meeting of the Guy's Hospital Physical Society next week the chair is to be taken, as it has been in many a year past, by Sir Samuel Wilks, who after eight-three years and holding many offices in the University of London, on royal commissions, on the General Medical Council, in addition to the physicianship at Guy's Hospital and the onerous duties of president of the Royal College of Physicians, is still hale and hearty, and well able to fulfil the part of chairman at these annual functions. The speaker of the evening is to be Dr. G. A. Gibson, the well-known Edinburgh physician, for some years editor of the *Edinburgh Medical Journal.*

The appearance of new journals reminds one of Dickens's description of a snowstorm: "It snowed, and snowed, and still it kept on snowing." And still new journals come: the latest which is just struggling to the birth is the *Quarterly Journal of Medicine,* edited by Professor Osler and a group of well-known collaborators. It is to be devoted to the publication of original papers dealing with clinical medicine, and will include critical reviews on special subjects and diseases. Although it is not formally stated, it is to be presumed that this new journal is intended to bear much the same relation to the recently formed Association of Physicians of Great Britain and Ireland as the journal *Brain* does to the Neurological Society; it will be largely a medium of publication for the subjects brought forward at the yearly meeting of the Association, but will not exclude original papers from other sources.

It is satisfactory to note that at University College Hospital honor is to be paid to the memory of two members of the staff who have recently passed away—Mr. Christopher Heath, and Dr. G. Vivian Poore. The former was Emeritus Professor of Clinical Surgery; the latter was Emeritus Professor of Medicine. Both were familiar figures to us who are still in active service at the medical schools of London, and both were men who commanded love as well as respect; and in these days, when there seems to be so little appreciation of true worth, it is a rare but pleasing event, the unveiling of medallion portraits in the Entrance Hall of University College Hospital by the president of the Royal College of Physicians. Sir William Douglas Powell, next week.